"EVALUATION OF EFFECTIVENESS OF PROPHYLACTIC PARENTERAL TRANEXEMIC ACID IN REDUCING BLOOD LOSS DURING AND AFTER ELECTIVE LOWER SEGMENT CESAREAN SECTION"

COIMBATORE MEDICAL COLLEGE

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In

OBSTETRICS AND GYNAECOLOGY

THE TAMILNADU

DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL 2016

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled **"EVALUATION OF EFFECTIVENESS OF PROPHYLACTIC PARENTERAL TRANEXEMIC ACID IN REDUCING BLOOD LOSS DURING AND AFTER ELECTIVE LOWER SEGMENT CESAREAN SECTION"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. P. SUNDARI** M.D.,DGO.,, Professor, Department of Obstetrics & Gynecology, Coimbatore medical college during the academic year 2013 - 2016.

This dissertation is submitted to **The Tamil Nadu Dr.M.G.R. Medical University,** towards the partial fulfillment of requirement for the award of M.S.Degree in Obstetrics and gynaecology.

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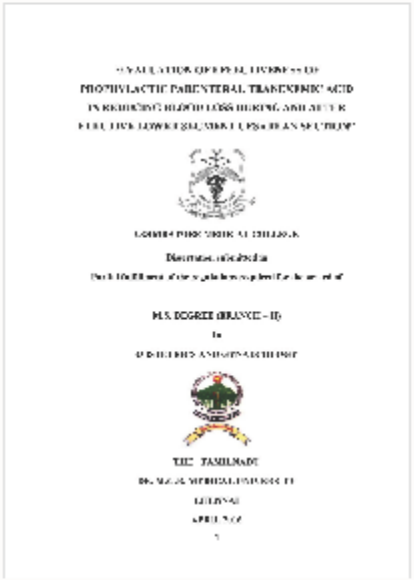

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CONTENTS

LIST OF ABBREVATIONS USED

ARR	-	Absolute risk reduction
BL	-	Blood loss
BT	-	Bleeding time
CI	-	Confidence interval
Cm	-	Centimeter
CS	-	Cesarean section
CT	-	Clotting time
EACA	-	Epsilon aminocaproic acid
Gm	-	Gram
Hb	-	Hemoglobin
Hct	-	Hematocrit
L	-	Litre
LSCS	-	Lower segment cesarean section
Mg	-	Milligram
min	-	minutes

Ml	-	milliliter
Pt	-	Patient
RR	-	Relative ratio
t-PA	-	Tissue plasminogen activator
TXA	-	Tranexamic acid

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ABSTRACT

BACKGROUND

Obstetric blood loss is the major cause of maternal mortality and cesarean section accounts for 25 -30 % of all deliveries. Delivery by cesarean section is associated with primary and secondary hemorrhage. Tranexemic acid is a synthetic derivative of amino acid lysine that exerts its anti fibrinolytic effects by its reversible blockade of lysine binding sites on plasminogen molecules.

Tranexemic acid is highly effective in reducing blood loss and hence the need for blood transfusion is also reduced in almost all kinds of surgery.

OBJECTIVES

Main objective is to study the effectiveness of prophylactic parenteral tranexemic acid in reducing blood loss during and after elective lower segment cesarean section.

METHODS:

A prospective randomized case control study conducted from August 2014 to August 2015 on 150 women who underwent lower segment cesarean section .75 of the cases were given tranexemic acid

before 20 minutes of skin incision. Other 75 subjects were not given tranexemic acid. The amount of blood loss from the end of placental delivery to two hours post partum was measured along with the vital parameters and adverse effects were compared among both the groups.

RESULTS

Tranexemic acid significantly reduces the amount of blood loss from the end of placental delivery to two hours post partum. In the present study it was observed that blood loss from the end of placental delivery to the end of surgery is 300 ml in study group and 380 ml in control group. In similar manner the amount of blood loss from the end of surgery to two hours post partum is 40 ml in study group and 70 ml in the control group with p value of <0.001 which is satistically significant. No complications or side effects are reported among both the groups.

CONCLUSION

Tranexemic acid significantly reduces the amount of blood loss during and after cesarean section and it was not associated with any significant side effects. Tranexemic acid can be used effectively in all women undergoing lower segment cesarean section.

INTRODUCTION

Obstetric blood loss is a major cause of maternal mortality and is always under estimated and hence blood is inadequately replaced. Caesarean section rate has been increasing nowadays accounting for 25 to 30 % of all deliveries[1] Caesarean section is specially associated with varying amount of blood loss. Though active management of third stage of labour is practiced well and it can prevent 60 %of post partum hemorrhage but still PPH has a devastating impact on the society with increased maternal mortality and morbidity.

The importance of tranexemic acid is that as it reduces blood loss as well as reduce the need of blood transfusion .Blood is a precious product in which patients are associated with risk of transfusion related adverse effects such as febrile non hemolytic transfusion reactions and blood borne infections.[2]

Tranexemic acid is a synthetic derivative of amino acid lysine that exerts its anti fibrinolytic effect through reversible blockade of lysine binding sites on plasminogen molecules.[3,4] Intravenous administration of tranexemic acid before skin incision routinely decreases the amount of blood loss during any surgery and also reduces the incidence of blood transfusion during surgery.

Tranexemic acid potentiates the blood clotting system as it is an anti fibrinolytic agent and is used to treat and prevent bleeding. During placental delivery rapid degradation of fibrinogen and fibrin occurs, as well as an increase in the activation of plasminogen activity and fibrin degradation products due to activation of the fibrinolytic system .This activation lasts for 6 to 10 hours post partum which may cause more hemorrhage .The anti fibrinolytic effect of tranexemic acid in the third stage of labour could make it safe and effective alternative or can be used as an adjunct to other regimen in the third stage of labour for prevention of post partum hemorrhage.[5]

In this present study the effectiveness of prophylactic tranexemic acid in reducing the blood loss from the end of placental delivery to two hours post partum in the third stage of labour during caesarean section has been investigated.

OBJECTIVE

The main objective of the study is to evaluate the effectiveness of tranexemic acid which when used prophylactically before skin incision is highly effective in reducing blood loss during and after elective lower segment ceasarean section.

REVIEW OF LITERATURE

Blood has two remarkable properties. It remains in the fluid state in the blood vessels throughout life, but rapidly becomes jelly like when shed. Both these properties are essential for the preservation of life. The latter property of blood that is losing its fluidity and setting into a semisolid jelly when shed or when collected in a test tube is clotting or coagulation of blood.

When the process of coagulation is studied under an electron microscope, it is seen that minute granules appear at first, often near a clump of disintegrating platelets. These granules join together to form a tough resilient meshwork of fibrils in which the cellular elements of blood get entangled as in a net. These fibrils are composed of fibrin.

Coagulation is the property of plasma alone ; the red and white corpuscles do not take part in it. They only become caught up in the meshes of the clot giving the characteristic appearance of a clot. It is due to the incorporation of the red cells that the clot appears red. When the clot subsequently contracts, a clear straw - colored fluid, called serum, is left behind. This will not clot any more.

The essential reaction in the conversion of the soluble hydrosol form of protein (fibrinogen) into an insoluble hydrogen form (fibrin)

by means of an enzyme thrombin. While fibrinogen exists in the circulating blood as such, thrombin is formed from an inactive circulating precursor called prothrombin, from the blood which is shed. The activation of prothrombin is brought about by the formation of prothrombin activators through a number of reactions of coagulation factors in the plasma, and from damaged tissue and from platelets.

Numerous theories have been proposed to account for the clotting of blood and a complicated and often confused array of terminology had sprung up in this field. To clarify the situation and to bring about a certain order, an international committee, which included the world's leading authorities on blood clotting was appointed.

They recommended that roman numerals should be used to describe the factors and gave a list of accepted factors as follows:

FACTOR I	**--**	**FIBRINOGEN**
FACTOR II	**--**	**PROTHROMBIN**
FACTOR III	**--**	**TISSUE THROMBOPLASTIN**
FACTOR IV	**–**	**CALCIUM**
FACTOR V	**–**	**LABILE FACTOR, ACCELERATOR GLOBULIN**
FACTOR VI	**--**	**UNASSIGNED FACTOR**
FACTOR VII	**--**	**STABLE FACTOR /SERUM PROTHROMBIN CONVERSION ACCELERATOR**
FACTOR VIII	**–**	**ANTI HAEMOPHILIC GLOBULIN**
FACTOR IX	**--**	**CHRISTMAS FACTOR**
FACTOR X	**--**	**STUART PROWER FACTOR**
FACTOR XI	**–**	**PLASMA THROMBOPLASTIN ANTECEDENT**
FACTOR XII	**–**	**HAGEMAN FACTOR/ GLASSS FACTOR**
FACTOR XIII	**–**	**FIBRIN STABILIZING FACTOR.**

However, they agreed that the first four factors could be called by their names, as these names are well established. The activated form of the factor is indicated by the subscript 'a' after the number.

BLOOD CLOTTING FACTORS

FIBRINOGEN

Fibrinogen is a plasma globulin formed in the liver. It is a dimer having two sets (A&B) of three linked polypeptide chains. Clot is formed by the conversion of fibrinogen into fibrin by the action of thrombin. Only fluids containing fibrinogen can clot. Hence plasma can clot but not serum.

PROTHROMBIN

Prothrombin is a plasma globulin formed in the liver and requires vitamin K. It is a monomer and is a proenzyme and a precursor of thrombin. Its plasma concentration is about 15 mg /100 ml and its half-life is about 3-4 days. It is absent in the serum.

THROMBIN

Thrombin is not normally present in the plasma. It is formed from prothrombin by the action of prothrombin activators in the presence of Ca^{++} ions .It is a protein with a molecular weight of

about 34000. It enzymatically converts fibrinogen to fibrin. It can also activate factor V & VII, and promotes platelet aggregation and activation.

TISSUE THROMBOPLASTIN

Tissue thromboplastin is released by injured tissues and is composed of tissue factor (a proteolytic enzyme) and tissue phospholipid. It activates factor VII. Thromboplastin is liberated from all tissues, but the most potent thromboplastin are found in extracts of brain , lung and placenta.

CALCIUM

Ionic calcium is essential for clotting of blood .Decalcified blood does not clot . Calcium ions are necessary for the activation of factors IX and X. Formation of prothrombin activators , and for activation of factor XIII.

FACTOR V (Labile factor, Accelerator globulin)

It is a heat labile high molecular weight protein formed in the liver. It is activated by factor X and thrombin , and is a co factor in the formation of prothrombin activator . It is used up during clotting and is absent in the serum. Its plasma half-life is about 36 hours.

FACTOR VII (Stable factor , or Serum Prothrombin Conversion Accelerator SPCA)

It is a protein formed in the liver and requires Vitamin K for synthesis. It is activated by tissue thromboplastin , and is a proenzyme for the activation of factor X. Its plasma half-life is 4 - 6 hours.

FACTOR VIII (Antihaemophilic globulin AHG, or AH factor A)

It is a high molecular weight globulin formed in the liver. It is transported in the blood combined with another protein von willebrand Factor (vWF) which is formed in the vascular endothelium (and also in the megakaryocytes). Factor VIII is activated on separation from vWF by thrombin and is a cofactor in the intrinsic pathway. It is used up during clotting and is absent in the serum. Its plasma life is about 12hours.Its deficiency causes Hemophilia A (vWF can also favor platelet adhesion)

FACTOR IX (Christmas factor, AH factor Plasma thromboplastin component PTC)

It is a protein formed in the liver and requires Vitamin K .It is activated by factor XI a and is a pro enzyme in the intrinsic pathway. Its Plasma half-life is about 24 hours. It is not used up in

clotting and is present in serum. It is called Christmas factor , because this factor was first found to be deficient in a patient named Christmas. Deficiency of this factor causes Christmas disease or Hemophilia B.

FACTOR X (Stuart prower factor)

It is a two chain protein formed in the liver and requires Vitamin.K.

It is a pro enzyme for the formation of prothrombin activators in both intrinsic and extrinsic coagulation pathways. It is activated by active factors VIII and IX in the intrinsic pathway and by factor VII a in the extrinsic pathway . Its plasma half-life is about 24 to 40 hours. It is present in the serum.

FACTOR XI (Plasma thromboplastin antecedent PTA or AH factorC)

It is a pro enzyme in the intrinsic pathway, which activates factor IX and is activated by factor XII and by HMW kininogen. This protein is formed in the liver. Its plasma half-life is 2 to 3 days, and is present in the serum.

FACTOR XII (Hageman factor, Glass factor)

It is a protein formed in the liver. Its plasma half-life is about 2 -3 days and it is present in the serum. It is activated by high molecular weight kininogen and prekallikrein , and by contact with sub endothelial collagen in the injured vessel. (Contact with negatively charged glass surface also activates factor 12).Activated XII converts pre kallikrein to kallikrein, which further activates factor XII . Activated factor XII is an intact pro enzyme which activates factor XI along with HMWK.Activation of factor XII is the first reaction that initiates the process of coagulation, as observed in test tube. However factor XII deficiency does not result in a clinical bleeding disorder, whereas factor XI deficiency does. Hence Factor XI activation must be considered to be an important initial reaction and there is apparently some mechanism in addition to factor XII, involved in activation of factor XI.

FACTOR XIII (Fibrin stabilizing factor Laki- Lorand factor)

It is a protein with 2 alpha and 2 beta chains with a plasma half-life 5 -7days. It is formed in the liver and probably megakaryocytes. It is activated by thrombin in the presence of Ca 2++ by forming cross linkage between the fibrin monomers and polymers.

PREKALLIKREIN (Pre-K, Fletcher factor)

It is a globulin and is a part of the kinin system. It activates factor XII which in turn activates prekallikrein to kallikrein .This pro enzyme is also involved in the activation of factor XI

HIGH MOLECULAR WEIGHT KININOGEN: (Fitzgerald factor)

It is an alpha globulin and is a cofactor in the activation of factor XI and XII.

PLATELET PHOSPHOLIPID

Injury to a blood vessel causes platelet aggregation as well as release from damaged platelets , platelet phospholipids which is a cofactor in the conversion of prothrombin to thrombin.

THE MECHANISM OF COAGULATION

The formation of a clot involves three basic steps. There are

1. Formation of prothrombin activators through two pathways
    a) Intrinsic pathway
    b) Extrinsic pathway
2. Formation of thrombin
3. Formation of fibrin

1. FORMATIONOF PROTHROMBIN ACTIVATORS

a) INTRINSIC PATHWAY

It is a series of cascading reactions that set up by trauma to blood, with contact of blood with sub endothelial collagen , and the release of phospholipid from platelets.

The first reaction is the activation of factor XII to XII a with the help of pro enzyme Pre –K and Cofactor HMWK. XIIa itself activates Pre -K which further increases XIIa formation.

1. XIIa with HMWK and Pre –K convert XI to XIa.
2. Now XI a in the presence of Ca^{++} activates IX to IXa.
3. Factor VIII is activated to VIII a by thrombin.
4. IXa along with cofactors VIIIa and phospholipid ,in the presence of Ca^{++} converts X to Xa.
5. Factor V is activated to Va by thrombin. Xa with co factors Va and phospholipids in the presence of Ca^{++} is the prothrombin activators which converts prothrombin to thrombin.

b) EXTRINSIC PATHWAY

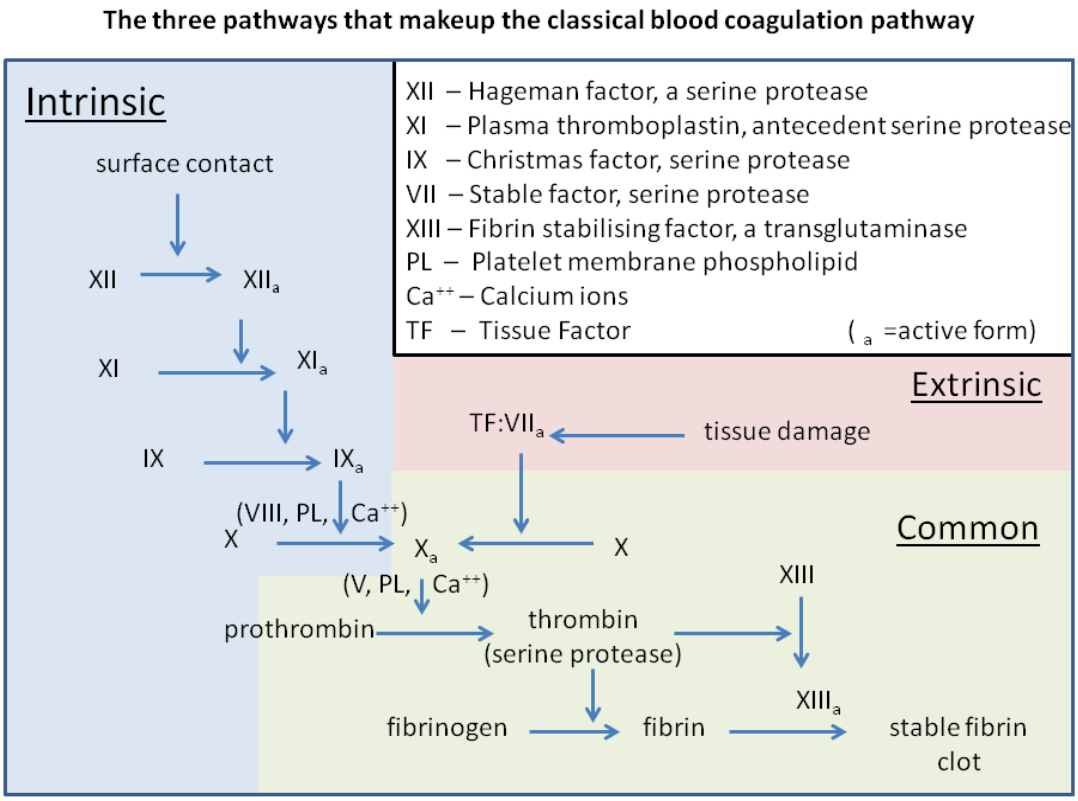
1. The process is initiated by trauma to tissue , which causes the release of tissue factor and tissue phospholipids (tissue thromboplastin) from injured tissues.
2. Tissue thromboplastin activates factor VII to VIIa which along with tissue phospholipid in the presence of Ca^{++} activates factor X to X a.
3. Activates X (Xa) along with co factors Va and phospholipids and Ca^{++} is the prothrombin activator.

2. FORMATION OF THROMBIN

The prothrombin activator with cofactors Va and phospholipids in the presence of Ca^{++} converts prothrombin to thrombin. As soon as prothrombin activator is formed and small amounts of thrombin are formed , the process of clot formation is accelerated as thrombin activates many of the clotting factors. The time taken for clot formation is due to the time taken for formation of prothrombin activator, especially via the intrinsic pathway which may take a few minutes. The extrinsic pathway is more rapid and may occur within a minute.

3. FORMATION OF FIBRIN (CLOT)

1. Thrombin converts fibrinogen to fibrin. It removes small peptides from the alpha and beta chains of fibrinogen to form fibrin monomers which soon polymerizes and form fibrin polymer.
2. Thrombin acts on the fibrin stabilizing factor XIII and activates it to XIIIa. This is an enzyme (transglutaminase),which in the presence of Ca^{++} sets up cross linkages between the fibrin fibers to form cross linked fibrin or stable fibrin.
3. The fibrin fibers form a network of delicate thread like fibrils in which RBC,WBC,Platelets and plasma are entrapped.
4. The clot becomes attached to the opening in the injured vessel, as the fibrin fibers become adherent to the wall of the vessel in the injured region. This helps to prevent further bleeding.



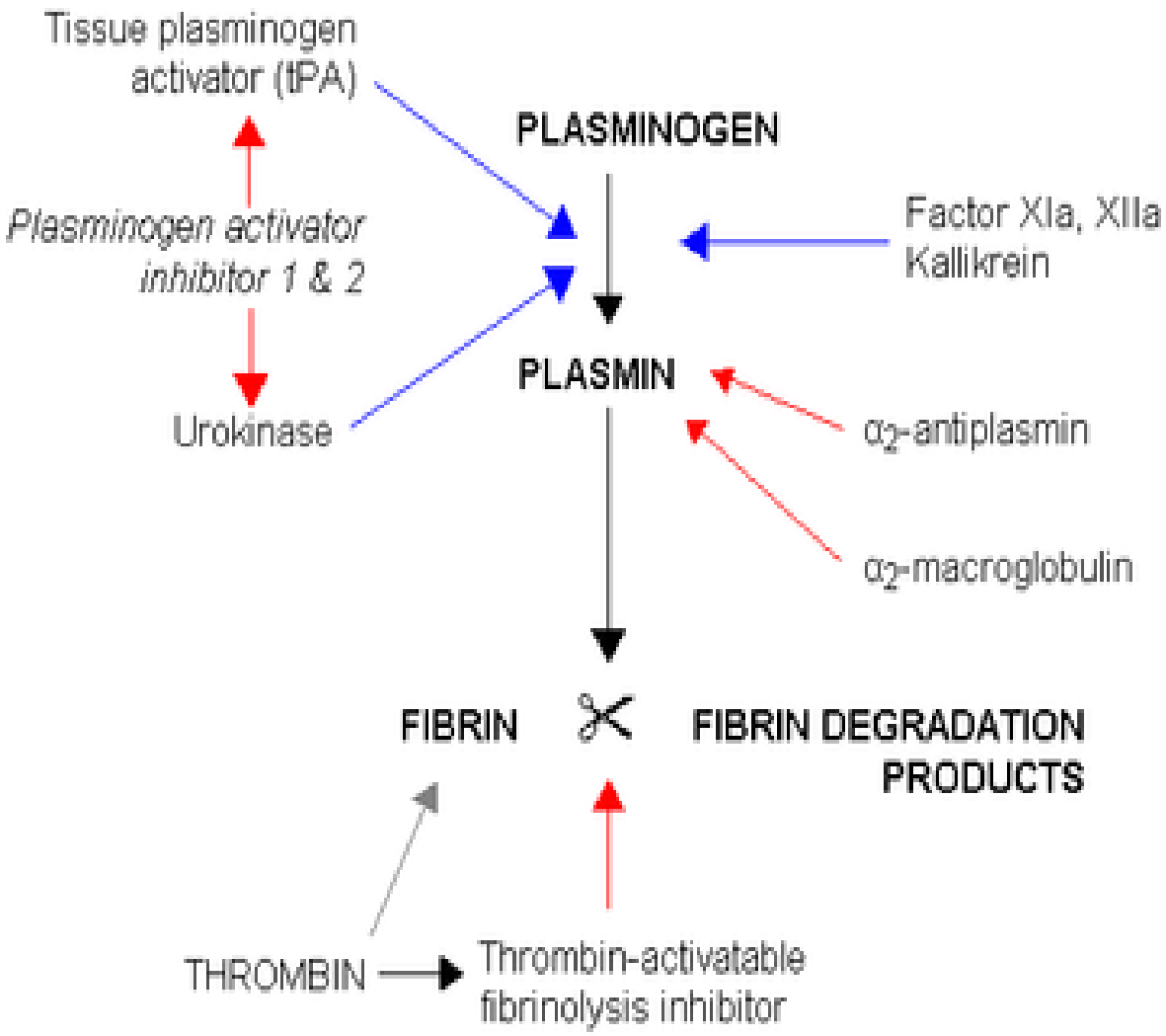
FIGURES.1 BLOOD COAGULATION PATHWAY

LYSIS OF CLOT (Degradation of Fibrin, Fibrinolysis)

In a day or two after clot formation and cessation of bleeding, the process of degradation of clot begins. The lysis of the clot is brought about by the action of a proteolytic enzyme called plasmin . Plasmin is not normally present in the plasma,but is formed from its precursor plasminogen which is a globulin formed in the liver and present in the plasma .Activation of plasminogen is brought about by plasminogen activators formed in the vascular endothelial cells.

The sequence of events is as follows:

1. Release of plasminogen activators. The formation and deposition of fibrin in the vessel wall (and probably the thrombin formed as well) stimulates the production of two plasminogen activators
   a) tissue type (t- PA)
   b) urokinase type (u – PA) from the endothelial cells
2. The t-PA and u-PA, which are protease enzyme act on plasminogen trapped in the clot and convert it into plasmin
3. The plasmin acts on the fibrin and fibrinogen and breaks them down to smaller soluble products called fibrin degradation products. Plasmin also digests the products and thus dissolves and removes the clot slowly in a few days. The process of fibrinolysis is regulated by controlling the activity of plasminogen activators. This is done by plasminogen activator inhibitors, which are also released by vascular endothelial cells. The plasmin that enters the plasma , or small amounts formed in the plasma is promptly inactivated by the alpha -2 antiplasmin, which is formed in the liver and is present in small concentration in the plasma.



FIGURES.2 MECHANISM OF FIBRINOLYSIS

FACTORS PREVENTING INTRAVASCULAR COAGULATION

Apart from smoothness of the vascular endothelium which prevents contact with collagen , there are substances such as protease inhibitors and other which prevent clot formation in the uninjured vascular system .

Some of these are:

1. Anti-thrombin 3 which is a protease inhibitor. It is a globulin .It links to heparin present in the blood and the complex formed becomes a potent anticoagulant ,and acts by inactivating the active forms of factors IX, X, XI, XII .It also binds with the thrombin that has not been absorbed to fibrin fibers , and this inhibits the effects of thrombin on fibrinogen.
2. Thrombomodulin is a protein formed by the capillary endothelial cells , and is present bound to endothelial membrane. It forms a complex with thrombin. The thrombomodulin - thrombin complex activates protein C,in the presence of cofactor protein S to form activated protein C. The APC inactivates factors VIII a and Va. Protein C and Protein S are formed in the liver, in the presence of Vitamin K and are present in the plasma. The binding of thrombomodulin to thrombin complex also favors fibrinolysis by inhibiting the inhibitors of plasminogen activators and increasing plasmin formation.
3. alpha 2 macroglobulin ,which is a protease inhibitor which is present on the endothelial surface also prevents activation of several coagulation factors of the intrinsic pathway and

appears to act in a manner similar to anti thrombin 3-heparin complex, except that it does not need heparin for activation. It also combines with thrombin and limits its action on fibrinogen.

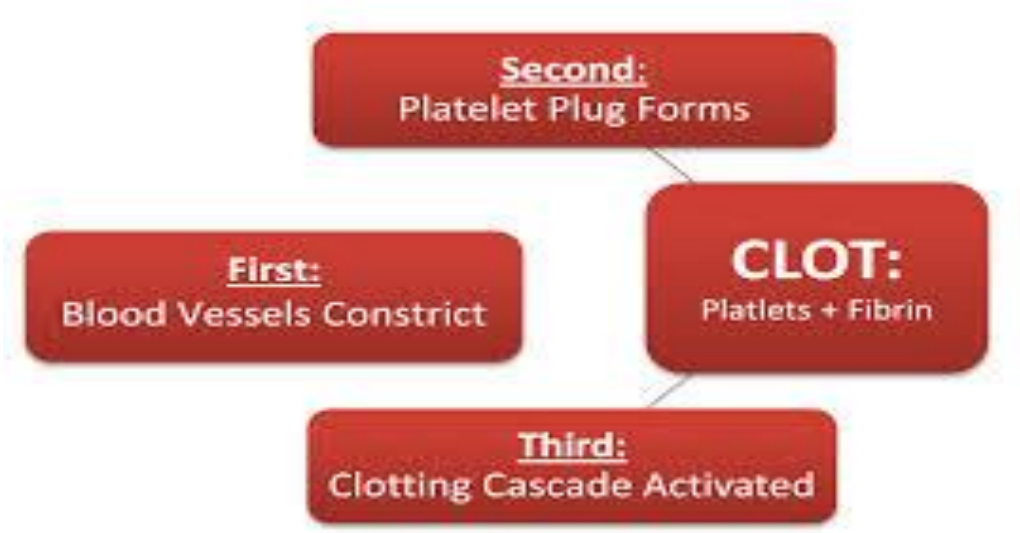
4. Extrinsic pathway Inhibitor: A new factor which causes inhibition of the extrinsic pathway has been recently identified. This is referred to as extrinsic pathway inhibitor or lipoprotein associated coagulation inhibitor. The Extrinsic pathway inhibitor along with Xa inhibits the enzymatic activity of factor VIIa / Tissue factor complex.

HAEMOSTASIS

Homeostasis is arrest of bleeding. The process of coagulation of blood takes 5 minutes to occur and is the secondary mechanism. . The immediate (primary) mechanism which occurs within seconds is

a) vasoconstriction at site of injury

b) Platelet plugs formation which seals the opening in the injured vessels.



FIGURES.3 MECHANISM OF HEMOSTASIS

VASOCONSTRICTION

Immediately after injury blood vessel constricts. This is largely due to the local response of vascular smooth muscle to trauma causing the muscles to powerfully contract. The contraction may last for a few minutes and is probably aided by release of some vasoconstrictor substances from the platelets. Even in large blood vessels like the radical artery may be fully constricted for a while. This immediate response helps prevent excessive bleeding from the site of injury.

FORMATION OF PLATELET PLUG

This process begins within a few seconds after injury and involves three mechanisms

a) Platelet Adhesion

b) Platelet Activation

c) Platelet Aggregation

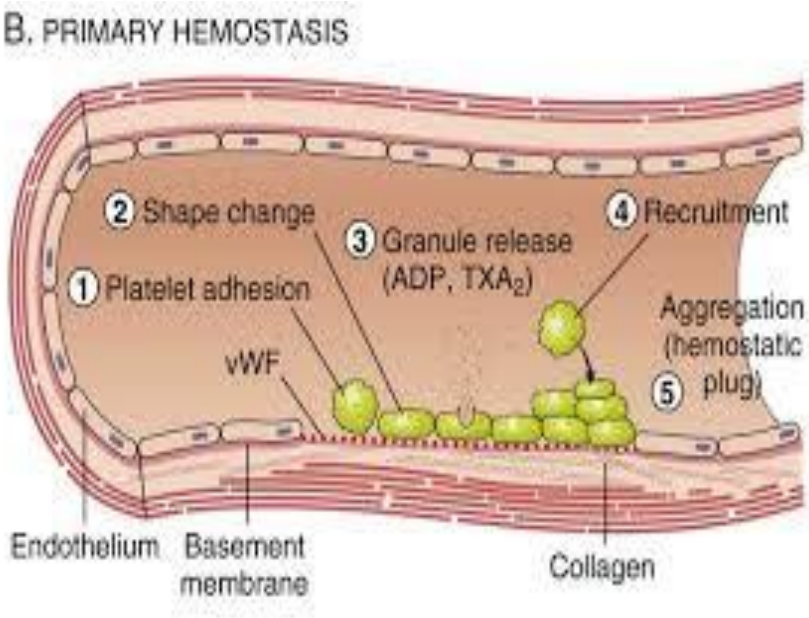


FIGURE.4 PRIMARY HEMOSTASIS

a) **PLATELET ADHESION**

When a blood vessel is injured, its sub endothelial collagen is exposed, and platelets adhere to the collagen. This process is assisted by the von Willebrand factor (vWF) which is present in the platelets as well as in endothelial cells.

b) PLATELET ACTIVATION

This process is initiated by the adherence of platelets to collagen and is facilitated by thrombin, thromboxane and platelet

activating factor which is a cytokine secreted by platelets as well as neutrophils and monocyte .The activated platelets swell change shape throw out pseudopodia and release the contents in their granules.

c) PLATELETS AGGREGATION

The secretion causes the platelets to get sticky and adhere to one another. The activation of the platelets draws more platelets towards them and activates them and these platelets attach to the collagen. As aggregation occurs the mass of platelets grow in size and form a haemostatic plug which may be quite effective in sealing off small opening in blood vessels. Integrins which bind to receptors, are one type of cell binding proteins called Cell Adhesion Molecule .Integrins are considered to play a main role in platelet adhesion and aggregation.

During day to day activities one frequently sustains minor trauma which is liable to cause bleeding from capillaries. It is the prompt formation of platelets plug that seals off small openings in capillaries and small venules that prevent bleeding. In platelet deficiency small haemorrhages under the skin and other parts

occur. Platelets also have a role in coagulation - eg : formation of platelet phospholipids.

The events that follows an injury to a blood vessel are

1. Vasoconstriction
2. Platelet plug formation
3. Blood coagulation
4. Clot retraction
5. Fibrinolysis

The processes are integrated with one another. The first two forms the primary haemostasis and the third secondary haemostasis.

COAGULATION IN PREGNANCY

Placental separation that occurs during the third stage of labour is the major haemostatic challenge in pregnancy. Myometrial contractions brings about significant constriction of blood vessels in the placental bed but adequate fibrin formation is also required.

Many physiological adaptation takes place during pregnancy and the haemostatic challenge is met in almost all pregnant women as pregnancy is always represented as a hypercoagulable

state. The concentration of many clotting factors increases during pregnancy which includes factor II ,VII, VIII, IX, XII. The concentration of clotting factors that tend to decrease during pregnancy is that of factor XI and XIII which occurs mainly during the third trimester of pregnancy.

TRANEXEMIC ACID

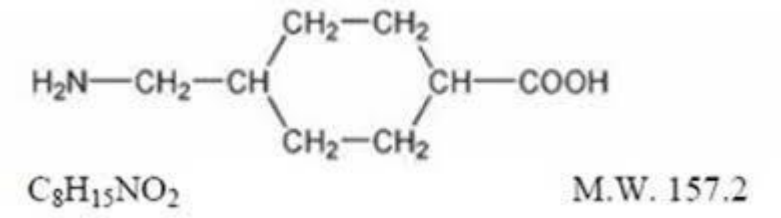


FIGURE 5 CHEMICAL STRUCTURE OF TRANEXEMIC ACID

Tranexemic acid is a synthetic lysine aminoacid derivative, which literally diminishes the dissolution of hemostasic fibrin by plasmin by exerting its anti fibrinolytic effects by reversible blockade of lysine binding site of plasminogen molecules.[7,8]

Thus the lysine receptors binding sites of plasmin for fibrin are well occupied by tranexemic acid thus preventing binding by fibrin molecule and hence preserving and stabilizing fibrin matrix.

The anti fibrinolytic effect of tranexemic acid is mainly mediated by reversible interaction that occurs at multiple binding sites of plasminogen. Actually human plasminogen contains 4 to 5 lysine binding sites which has low affinity towards tranexemic acid and only one high affinity sites. This high affinity site is mainly involved in its binding with fibrin saturation of this high affinity binding site with that of tranexemic acid leads to displacement of plasminogen from its surface of fibrin . Inspite of the fact that plasmin may be formed just by conformational changes in plasminogen, the process of binding to fibrin and dissolution of the fibrin is totally inhibited by tranexemic acid.[9]

FIGURE 6 TRANEXEMIC ACID INJECTION

PHARMACODYNAMICS

Tranexemic acid reduces 20 -60 % the rate of lysis of fibrin which is catalyzed by tissue plasminogen activators[10]. In all healthy individuals ,tranexemic acid when in concentration of 10 mg /ml in the blood has no effect on the platelet count as well as various coagulation factors and hence no change or effects on the coagulation time.

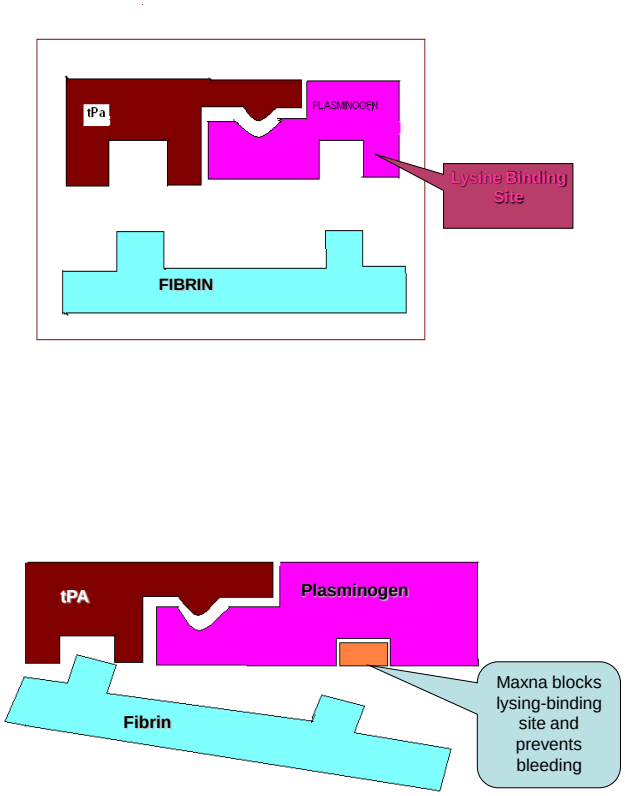


FIGURE 7 MECHANISM OF ACTION OF TRANEXEMIC ACID

PHARMACOKINETICS

ABSORPTION

After a single oral administration the peak concentration in plasma occurs approximately by 3 hours. Tranexemic acid can be administered without regards to meals.[11, 12.]

DISTRIBUTION

- Tranexemic acid is 3 % plasma bound with no apparent binding with albumin[10]
- Tranexemic acid crosses placenta , the concentration in cord blood is 30 mg/L after intravenous injection of 10 mg/kg of tranexemic acid in pregnant women.[13]
- Tranexemic acid gets one tenth concentration in cerebro spinal fluid as that in the plasma.[14]
- Tranexemic acid also passes into aqueous humor of eye and gets one tenth concentration as that in plasma.[15]

METABOLISM

Only a small fraction of the drug is metabolized[16]

EXCRETION

Tranexemic acid gets eliminated via urinary excretion that too primarily by glomerular filtration with more than 95 %of drug excreted unchanged. 90 % of drug get eliminated within24 hours of intravenous administration. After intravenous dosage most of drug gets eliminated during the first 10 hours followed by other fractions . Thus elimination half life is estimated to be 2 hours. [17 , 18.]

USES OF TRANEXEMIC ACID

CARDIAC SURGERY

In patients undergoing cardiac surgery mainly with cardio pulmonary bypass peri operative treatment of tranexemic acid as a intravenous loading dose results in significant reduction in blood loss that may occur post operatively.[19, 20]

A meta-analysis of nearly 60 randomized clinical trials in cardiopulmonary bypass showed that tranexemic acid is solely associated with a significant reduction in proportion of cardiac patients requiring blood transfusion peri operatively.[30 ,31, 32.]

ACUTE UPPER GASTRO INTESTINAL BLEEDING

Highly great statistical significant data results of reduction of blood transfusion requirement with that of tranexemic acid therapy with upper gastro intestinal bleeding patients has been obtained in many published trial.[33 ,36, 37.]

Meta analysis reveals that tranexemic acid reduces the risk of rebleeding by 30 % and 30 % to 40% reduction for need of surgery and last but not the least reduces mortality by 40 %[34 ,35.]

ORAL SURGERY

When tranexemic acid is used as a mouthwash after oral surgery [38] in patients receiving oral anticoagulants therapy ranges between 0 to 6.7 % [42, 43, 44.] only compared to the placebo where the rate ranges from 13.3 to 40 %[38 , 39, 40 ,41]

OTHER SURGERY

The list of use of tranexemic acid extends way ahead , it is significantly useful in the following surgeries.

- Total knee replacement[57, 58.]
- Orthotopic liver transplantation[59, 60 ,61]

GYNAECOLOGIST

Tranexemic acid plays a vital role in all gynaecologist practice. It is a effective method to control bleeding which may be either menorrhagia, peri operative loss.[23, 24, 25]

MENORRHAGIA

The main outpatient population comes commonly with complaints of excessive bleeding per vagina. During normal periods the amount of blood loss is approximately 30 -50 ml. if the blood loss is more than 80 ml ,it is defined as menorrhagia Many trials has been conducted which has resulted in nearly 50 % reduction of blood loss during menstruation.[21 ,22.]

BLEEDING ASSOCIATED WITH PREGNANCY

During pregnancy bleeding may occur due to

1. Postpartum hemorrhage
2. Abruptio placenta
3. Placenta previa

Various studies has demonstrated that usage of tranexemic acid has been associated with three to four fold increase in perinatal morbidity. In case of abruption placenta studies have

shown that perinatal mortality is reduced by 8 %. It also reduces the amount of blood loss in case of central placenta praevia who presents as obstetric emergency with that of heavy bleeding.[26,27]

Various randomized multicentric trials discuss significant reduction in bleeding after placental delivery to two hours postpartum with no associated side effects.[28 ,29.]

HEREDITARY ANGIONEUROTIC EDEMA[64, 65.]

Hereditary angioneurotic edema is mainly characterized by recurrent non pitting sub epithelial edema which may involve any part of body. In double blind studies conducted results in reduction of severity of attacks of edema when tranexemic acid is giving at a dose of 1.5 g/day for seven days after attack.[66 ,67, 68]

SUBARACHNOID HEMORRHAGE

Rupture of any of the intracranial aneurysm results in bleeding into the subarachnoid space. In a study where the patients who received anti fibrinolytic therapy as compared to those who did not receive the treatment, resulted in significant reduction in the rate of re-bleeding.[69, 70, 71, 72.]

USE IN TRAUMATIC HEMORRHAGE

In our body, almost hemostatic system maintains the integrity of circulating system after every vascular injury which may be traumatic or surgical in its origin.[45, 46, 47,48.] Major trauma or surgery brings about a trigger in the hemostatic system of the body or it may be even from the massive loss of blood from the body[53, 54, 55.]. All these provides the coagulation system of our body which is a major challenge. In this place anti fibrinolytic agent plays a major role in the reduction of blood loss without increasing any risks of post operative complications as well as no increase in the risk of venous thromboembolism. Many studies have been conducted which showed results has significantly reduced in the need for transfusion by one third as well as reduced the volume of transfusion by one unit and also has halved the need for further surgery in order to control bleeding[49]. These differences were all satistically significant as that anti fibrinolytic agent reduces blood loss need for transfusion and mortality. The blood is a scarce and expensive resourse and major concern regarding risk of transfusion transmitted infection, thus a simple and widely accepted practice of using anti

fibrinolytic agents that reduces blood loss following may be trauma or surgery and hence prevents thousands and thousands of death each year and it may also reduce exposure to all the risk of blood transfusion.[50]

The large trial namely **CRASH 2 . THE CLINICAL RANDOMIZATION OF AN ANTI FIBRINOLYTIC IN SIGNIFICANT HEMORRHAGE**[56] where the comparison between anti fibrinolytic and placebo which has significantly showed reduction in death as well as the need for transfusion in all those patients [51 ,52.]

ADVERSE DRUG REACTION

Along with all of its effects it may cause certain unwanted effects. The same effects of tranexemic acid that prevents bleeding may lead on the formation of clot that could be dangerous warrenting medical attention in such situation [73, 74, 75] . The possible signs and symptoms that may occur leads to

- Head ache
- Pain in chest, groin, calf
- Sudden loss of co - ordination
- Sudden slurred speech

- Sudden vision changes[76, 77, 78]
- Sudden shortness of breath
- Weakness or numbness in arm / leg

Other bothersome side effects which may be present are

- Nausea or vomiting
- Diarrhea
- Dry ejaculation and watery eyes[79, 80.]

DRUG INTERACTION

Still now ,no drug interaction has been reported[81 ,82, 83.]

OVERDOSE

No known case of over dosage of tranexemic acid is still reported. Symptoms of overdosage includes

- Nausea
- Vomiting
- Hypotension

CONTRAINDICATION

1. In patient with acquired defective colour vision as it prohibits measuring one end point which has to be followed as a measure of toxicity
2. In patients with sub arachanoid hemorrhage , sometimes cerebral edema and cerebral infarction may be caused by tranexemic acid.
3. Past history of thromboembolism episodes or with active intravascular clotting.
4. Heart disease, kidney disease, liver disease.

CAESAREAN SECTION

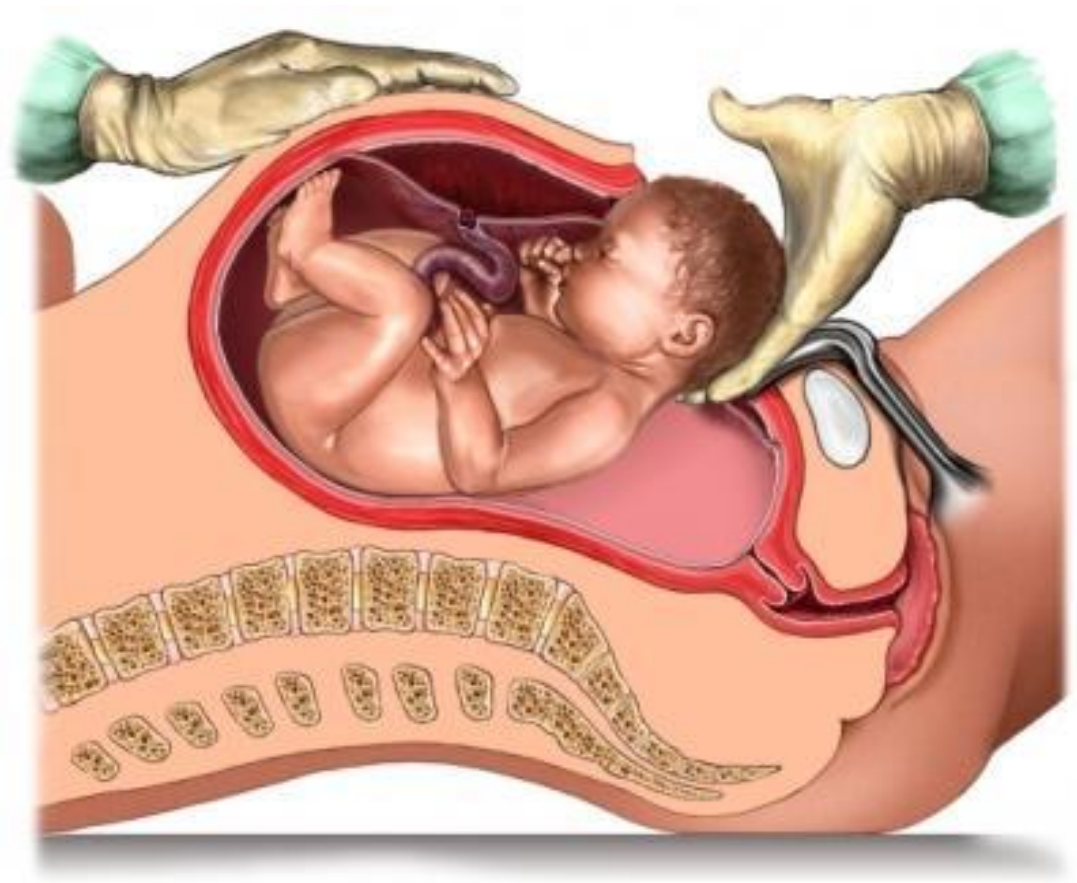


FIGURE 8 CESAREAN SECTION DELIVERY

Caesarean section is the most commonly performed operation now a days. There is a steady increase in the rate of ceasarean section. Ceaserean delivery is defined as the process of delivery of the foetus after the period of viability through an abdominal incision and uterine incision made in an intact uterus.

SURGICAL ANATOMY OF CEASAREAN SECTION DELIVERY

THE ABDOMINAL WALL

Knowledge about various abdominal wall layers are very much essential for the surgeon to enter into the abdominal cavity as well as to ensure maximum safety and efficiency. Thus the various abdominal wall layers includes

- Skin
- Subcutaneous layers
    - Camper's fascia
    - Scarpa's fascia
- Musculo aponeurotic layer
- Rectus sheath
- Rectus abdominus muscle
- External oblique muscle
- Internal oblique muscle
- Transverse abdominus muscle
- Transversalis fascia
- Peritoneum

BLOOD SUPPLY

SKIN

The skin near the midline is supplied by three main arteries

- Superior epigastric artery
- Inferior epigastric artery
- Superficial epigastric artery

Almost all superficial arteries accompany along with cutaneous nerves.

The superficial epigastric artery run from the femoral artery towards umbilicus in a diagonal course in the subcutaneous tissue.The skin along the flanks are supplied from the branches of the branches of following arteries

- Intercostal arteries
- Lumbar arteries
- Deep circumflex iliac vessels

VENOUS DRAINAGE OF SKIN

Venous system is the network of veins that radiate from the umbilicus. The veins above the umbilicus drains into the

lateral thoracic vein and axillary vein which in turn drains into superior vena cava.The veins below the umbilicus drains via superficial epigastric and great saphenous vein which in turn drains into femoral vein and ultimately into inferior vena cava.

ABDOMINAL WALL BLOOD SUPPLY

Main arterial blood supply include

1. Superior epigastric artery
2. Musculo phrenic artery
3. Deep circumflex artery
4. Inferior epigastric artery

The medial part of whole abdominal wall is supplied by epigastric vessels where as the lateral wall is supplied from the musculophrenic and deep circumflex iliac vessel along with lower intercostals and lumbar vessels.

The linea alba present in the midline where all fascia converge is relatively bloodless and hence we have to be very careful in case of low midline incision due to bloodless field, The healing rate is lowered.The superior epigastric artery which is the terminal branch of internal thoracic artery enters into rectus sheath where it descends behind rectus muscle and gets

anastomosed with that of inferior epigastric artery. It supplies the upper central part of the anterior abdominal wall layer .The inferior epigastric artery which is the branch of external iliac artery at the level just above to that of inguinal ligaments.

It enters the medial end of deep inguinal ring and runs upwards where it pierces the transversalis fascia and hence enters the rectus sheath just anterior to that of arcuate line.

It ascends and gets anastomosed with that of superior epigastric vessel.It supplies the lower central part of the anterior abdominal wall layers.

The deep circumflex iliac artery which is also a branch of the external iliac artery at the level of inguinal ligament runs upward and laterally towards anterior superior iliac spine continuing along with that of iliac crest.

It mainly supplies the lower later part of abdominal wall.The two posterior intercostals arteries, four lumbar arteries run between the muscle layers and supply the lateral part of the abdominal wall . The main vessel that is subjected to injury is that of epigastric artery that too particularly when the muscle splitting incision is made.

VENOUS DRAINAGE

The venous blood forms a network and drains above into the axillary vein and from below to the femoral veins. A few para umbilical vein through the ligamentum teres drain into the portal vein.The superior epigastric, inferior epigastric and deep circumflex veins drain into internal thoracic and external iliac veins.The posterior intercostals veins drain into azygos and lumbar veins drain directly into that of inferior vena cava.

BLOOD SUPPLY OF UTERUS

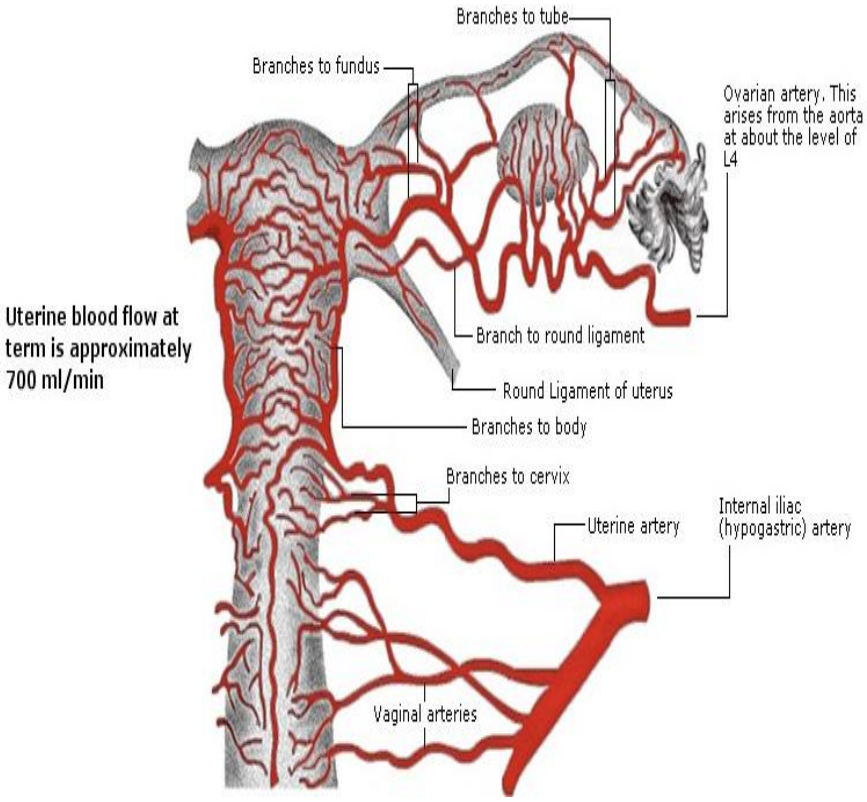


FIGURE. 9 BLOOD SUPPLY OF UTERUS

The main artery that is supplying uterus is the uterine artery. There are two uterine artery that is present on either side of the uterus and this uterine artery is ultimately the branch of anterior division of internal iliac vessel. It runs medially along the cervix crosses the ureter at the level above the lateral fornix that is 2 cm lateral to cervix and it ascends upwards along the lateral wall of uterus that between the two leaves of broad ligament and at the level of corneal end of the fallopian tube, it gets anastomosed with that of ovarian artery. The uterine artery give rise to anterior and posterior arcuate branches along the whole length of uterus it usually enter transversely into the myometrium of the uterus from where it gives branches as radial branches. These radial branches almost anastomosing that of their counterpart radial branches. These before reaching the endometrium tend to supply the basal layer of deciduas by its basal branch and make their entry into the deciduas by spiral arteries that are highly tortuous. The spiral artery reaches their maximum spiraling at 20 weeks. Later on as pregnancy passes these spiral arteries become straight and engorged so that the initial blood supply of uterus which was 50 ml/min increases to the level of 450 -600 ml /min at term. The uterine vein which is engorged in

pregnancy drain into internal iliac veins. The another blood supply is from the ovarian artery which arises from abdominal aorta below the level of renal artery. It crosses the ureter near its origin in retro peritoneal space and enters into the suspensory ligament of ovary .It courses along the underneath fallopian tube and gets anastomosed with that of uterine vessels at the level of cornua of the fallopian tube. The ovarian veins that is arising from pampiniform plexus at the level of ovarian hilum drains into inferior vena cava on right side and left renal vein on left side.

The main cause for blood loss during caesarean section includes

1) Injury to inferior epigastric vessels
2) Injury to uterine artery by lateral extension of the incision
3) Upper uterine segment incision
4) Manual removal of placenta

It is well documented that the use of tranexemic acid decreases the blood loss by 40 -50 %.

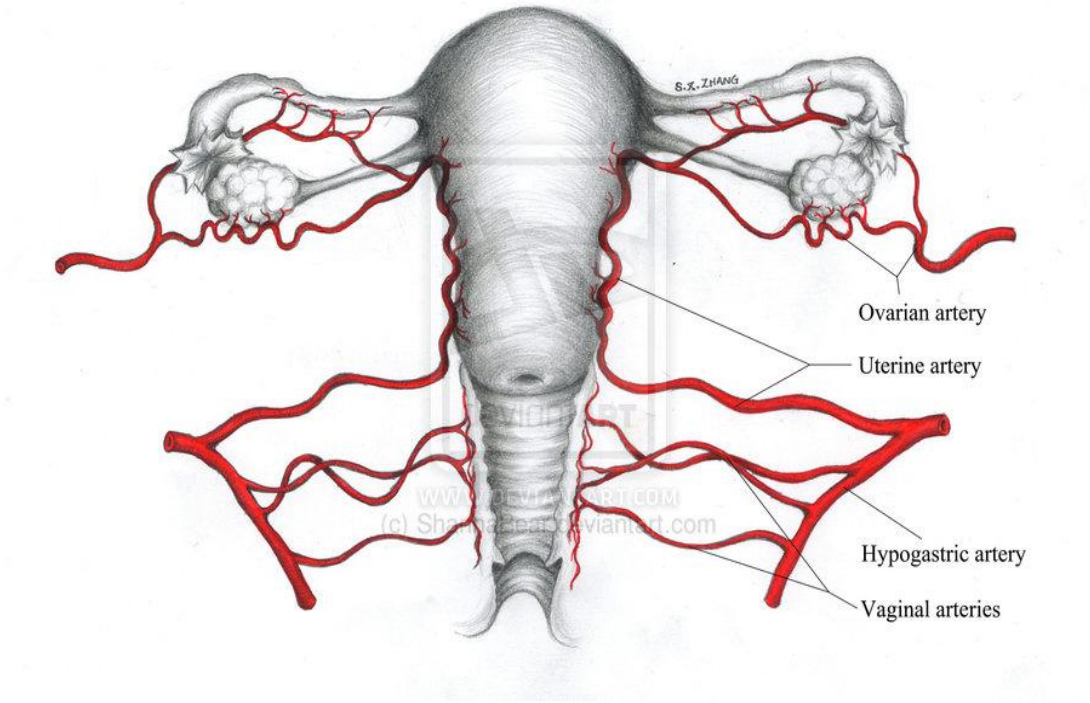


FIGURE. 9 BLOOD SUPPLY OF UTERUS

VARIOUS STUDIES

- Study conducted by Mayur et al has shown that usage of tranexemic acid has led to significant reduction in blood loss from the end of plancental delivery to that of two hours postpartum.[84]It showed that the amount of blood loss from the end of LSCS to two hours postpartum is 75.71 ml in study group versus 133.03 ml in that of control group. It also reduces the amount of blood loss from time of placental delivery to the end of LSCS as 372.71 ml in study group

where as it was 469.70 ml in control group. No obvious complications were reported.

- Another study that is conducted by Tastsumoti et al randomized case control trial where the quantity of blood loss was assessed in 180 primipara who are divided into control and study group. This also suggested decrease in the quantity of blood loss in the study group.
- Another study by Yang H et al includes dividing subjects into four groups as follows[85 ,86.]

Group 1: Tranexemic acid 1 gm given slow intravenously

Group 2: Tranexemic acid 0.5 gm given slow intravenously

Group 3:Aminomethyl benzoic acid 0.5 gm given intravenously

Group 4: No treatment

The amount of blood loss is calculated from the time of expulsion of placenta and two hours after delivery. The total amount of blood loss calculated were 243.3 ml,24.9ml, 308.1ml,314.8 ml respectively. The blood loss in group 1 and group 2 were significantly less compared to that of group 3 and group four. The incidence of post partum hemorrhage is also less in group 1 and group2. Still 1 gm of tranexemic acid is highly

efficacious and also safe in the aspect of reducing postpartum blood loss in these subjects.

- Afsar et al conducted a randomized study in 90 primipara where 45 women were given tranexemic acid and 45 women who are controls receiving placebo . The blood loss as well as hemotocrit are measured, it showed the blood loss in tranexemic acid group is 28.02ml and that of control group is that of 37.12 ml measured from the end of ceaserean section to two hours post partum. No complications or side effects was reported in either of the groups.
- Svanberget al studied the effect of intramuscular usage of tranexemic acid in 67 abruptio placenta patients and concluded that the routine use of tranexemic acid in patients with abruptio placenta can reduce the perinatal mortality.[87]

IN MENSTRUAL BLEEDING

- Lethalyet al concluded that usage of tranexemic acid in patients with heavy menstrual bleeding resulted in greater reduction in the bleeding compared with that of placebo significantly.[88 , 89.]

IN CARDIAC SURGERY

- Brown and morgan et al in randomized placebo controlled trial who are undergoing coronary artery bypass grafting surgery. [90.]Tranexemic acid is administerd before and during the operation showed they are highly efficacious in reducing the blood loss and need for blood transfusion.

GYNAECOLOGY SURGERY

- Caglar et al has studied effect of tranexemic acid in patients undergoing myomectomy .It has reduced peri operative and post operative bleeding as well as need of blood transfusion has decreased.[91, 92, 93,94.]

ORTHOPEDIC SURGERY

- Rajesparen et al shows the effect of tranexemic acid in total hip replacement it has reduced post operative blood loss. The treatment was not associated with any thromboembolic complication.[57,58.]

QUANTIFICATION OF BLOOD LOSS

Hemorrhage in obstetric accounts for about 25 % of all maternal deaths. PPH is defined as any bleeding in excesss of 500 ml after delivery. Although 60 % of all cases of PPH can be controlled by active management of third stage of labour . Obstetric hemorrhage has resulted in nearly 30 % of mortality or maternal death . The fifth millennium development goal to reduce maternal mortality to 75 % by 2015 cannot be attained without taking into consideration PPH in developing countries.Clinicians continue to rely on visual estimates to determine the amount of postpartum blood loss but often visual estimate are found to be inaccurate There are various methods that can be used in the estimate of blood loss during the postpartum period.[99]

VISUAL ASSESSMENT

The observation used in the visual estimate of blood is relatively straight forward and hence it requires no expenditure.

DIRECT AND STANDARD MEASURING JAR

This approach used by WHO in multicentric trial of usage of misoprostal in active management of third stage of labour .The collected blood was poured into standard measuring jar and its whole volume was measured.

RUBBERISED BLOOD MAT

In Bangladesh the international center for diarrhoeal disease research developed a rubberized blood mat which gets saturated with 500 ml of blood, with flowered plastic on one side and rubber on other side . When after delivery,the mat holds maximum of 500 ml of blood . After that the mat begins to leak into plastic and then on floor, which signifies post partum hemorrhage.

KELLY'S PAD

The continuum of care project which was implemented in India which is a rubberized mat called as kelly' pad in order to measure the blood loss. The kellys pad funnels the blood into a calibrated bowl where there is an alert line at about 500 ml. This pad is washable and sterilized and it is a cost effective method.

GRAVIMETRIC METHOD

This method involves weighing sponges before and after use. This method requires the weighing of all soaked pads on scale and to subtract the known weight of the other materials used inorder to determine the blood loss. Inaccuracy may arise at several levels due to lack of international standardization of size and weight of guaze,sponges and pads.[95,96,97,98.]

LABORATORY BASED METHOD

Alkaline hematin /acid hematin method:

In this the blood is added to a standardized solution which converts hemoglobin to acid hematin or cyanmethemoglobin. This can be measured by a spectrophotometer or calorimeter.

FIGURE.10 PICTORIAL REPRESENTATION OF BLOOD LOSS

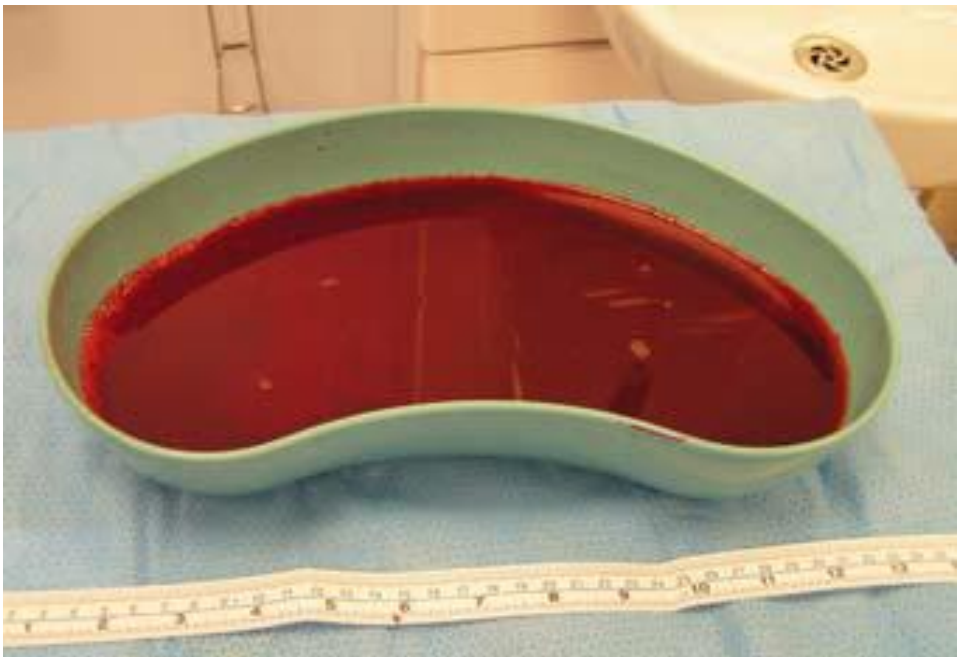
SOILED SANITARY TOWEL - 30 ML

SOAKED SANITARY PAD - 100ML

SMALL SOAKED SWAB 10 x10 cm -- 60ml

LARGE SOAKED SWAB 45x45cm ---350ml

FULL KIDNEY DISH --500ml



PPH ON BED ONLY -- 1000 ml

PPH SPILLING ON FLOOR -2000 ml

MAGNITUDE OF THE PROBLEM

The importance of knowing the amount of blood loss is that this is the leading cause of maternal mortality and morbidity, when we fail to recognize the excessive blood loss that occurs during child birth ,if early intervention are not initiated obstetric hemorrhage can lead to death which is the worst complication of pregnancy.

SUGGESTED EQUIPMENTS

Various equipments that are being suggested to measure blood loss include

1. Calibrated under buttock drapes
2. Scales to weigh the soaked items
3. Dry weight cards laminated and are attached to all scales for measurement of blood loss

Formulas inserted into electronic charting system which can automatically subtract the dry weight from that of wet weight in standard supplies like chux and peripads.

MATERIALS AND METHODS

PARTICIPANTS AND STUDY DESIGN

Pregnant women undergoing LSCS in Coimbatore Medical College Hospital. It is a prospective randomized case control study commencing from August 2014 to August 2015.

Sample size : 150.

They are allocated in two groups

1. STUDY GROUP: Subject who receive Tranexemic acid
2. CONTROL GROUP: Subject who did not receive Tranexemic acid.

DETAILS OF THE STUDY

INCLUSION CRITERIA

Full term primigravida or multigravida with singleton pregnancy delivered by LSCS.

EXCLUSION CRITERIA

1) Medical or surgical problem involving heart, liver, kidney, brain.
2) History of thrombo embolic disorder.
3) Pregnancy complications such as severe pre-eclampsia , abruption placenta, placentaprevia, fibroid complicating pregnancy.

4) Any blood dyscrasia

5) Allergy to Tranexemic acid.

6) Multiple pregnancy,polyhydramnios.

PARTICIPANTS AND STUDY DESIGN

Pregnant women undergoing caesarean section in Coimbatore medical college hospital .It is a prospective randomized case control study commencing from August 2014 to August 2015. Totally 150 subjects are taken for study according to the inclusion and exclusion criteria protocol.

ADMINISTRATION

Tranexemic acid is administered 15 to 20 minutes before incision in a dosage of one gram intravenous slowly infused over 5 minutes. After delivery of the neonate 10 units of oxytocin is administered in intravenous drip.

CONTROL GROUP

Tranexemic acid is not administered oxytocin 10 units is administered in intravenous drip after the delivery of the neonate.

OBSERVATIONS

CLINICAL OBSERVATION

1. VITAL SIGNS

Heart rate ,respiratory rate,blood pressure were checked immediately after placental delivery as well as one hour and two hour after birth of the neonate.

2. BLOOD LOSS

The blood loss was measured from the period of placental delivery to the end of surgery and then again from the end of operation to two hours postoperatively. The weight and volume of blood loss was estimated.

3. Assess the contraction of uterus and placental separation.
4. Neonatal outcome by apgar scoring
5. Any obvious side effects caused by administration of tranexemic acid.

LABORATORY EXAMINATION

1. Complete blood count with platelet count done before delivery and 24 hours after delivery.

2. Renal function test and liver function test is done before delivery and second day post operatively.
3. Coagulation profile is performed before and after delivery.

QUANTIFICATION OF BLOOD LOSS:

Blood was collected into the suction container after the placental separation, thus excluding the collection of amniotic fluid into the suction container, so that the collected blood is only from that has occurred after placental delivery.

All the soaked guage pads and operation table sheets are weighed. The measurement in two different period one from the time of placental separation to the end of surgery .Next one from the end of surgery to that of two hours postpartum.

Quantity of blood loss = (weight of used material + weight of unused material) - (weight of all material prior to surgery) + volume collected in the suction container after placental delivery.

Evaluation of effectiveness of tranexemic acid is given by

1. Efficacy of the drug
2. Safety of the drug.

EFFICACY :

Efficacy is given by

1. Amount of blood loss
2. The incidence of PPH

SAFETY:

Safety profile is given by

1. Monitoring vital signs
2. General side effects with administration of drug
3. Labarotory findings.

SAMPLE SIZE

Control group : 75 subjects

Study group : 75 subjects

Total : 150 subjects

They are allocated into two groups

1. STUDY GROUP : subjects who receive tranexemic acid
2. CONTROL GROUP :subject who did not receive tranexemic acid

All cases included in both groups are selected according to the inclusion and exclusion criteria protocol after getting consent.

RESULTS

The prospective ramdomised case control study conducted in Coimbatore medical college among 150 pregnant women undergoing elective lower segment caesarean section from the period of August 2014 to August 2015 were all included in the study.

From 150 subjects 75 received tranexemic acid and 75 did not receive tranexemic acid

TABLE 1

DISTRIBUTION BASED ON PATIENT CHARACTERISTICSIN THE STUDY

	STUDY GROUP (mean +/- SD) (n= 75)	**CONTROL GROUP (mean +/- SD) (n=75)**	**P value**
AGE	26.33 +/- 4.14	25.56 +/-3.85	0.238

Table 1. shows that the possible confounding variable age which is matched effectively in both groups of study and control.

Mean age was 26.33 in study group and 25.56 in control group with p value as 0.238. The difference in age of subject of both group is not satistically significant.

TABLE 2

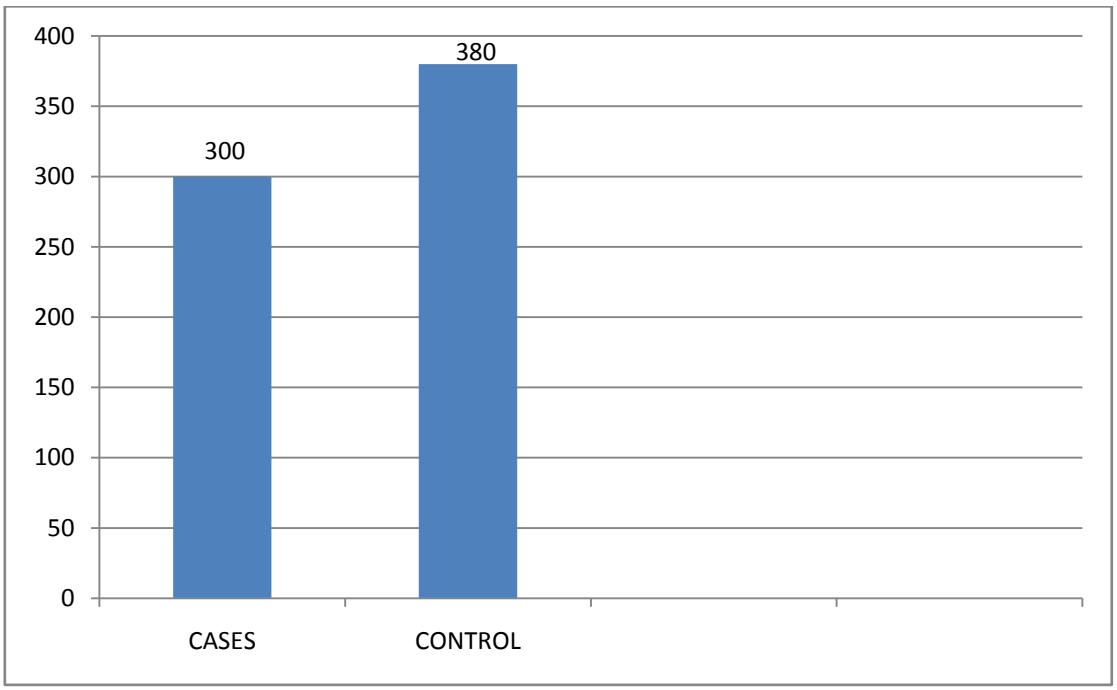
COMPARISON OF BLOOD LOSS FROM TIME OF PLACENTAL DELIVERY TO COMPLETION OF SKIN CLOSURE

BLOOD LOSS (ml)	**CASES**			**CONTROL**			**P value**
Placental delivery to end of surgery	maximum	minimum	Median	maximum	minimum	median	<0.001
	320	250	300	960	350	380	

Thus table 2 shows that the mean blood loss from time of placental delivery to completion of caesarean section ,there is a median loss of 300 ml whereas it is 380 ml in control group with p value of <0.001 which is statistically significant blood loss in both study and control group. Patients who received tranexemic acid has nearly 80 ml of blood loss less than those of patients who did not receive tranexemic acid.

GRAPH 1

BAR DIAGRAM SHOWING COMPARISON OF BLOOD LOSS FROM THE TIME OF PLACENTAL DELIVERY TO END OF SURGERY



Now coming on to comparison of blood loss from the end of caesarean section to two hours postpartum is as follows as given in the table

TABLE 3

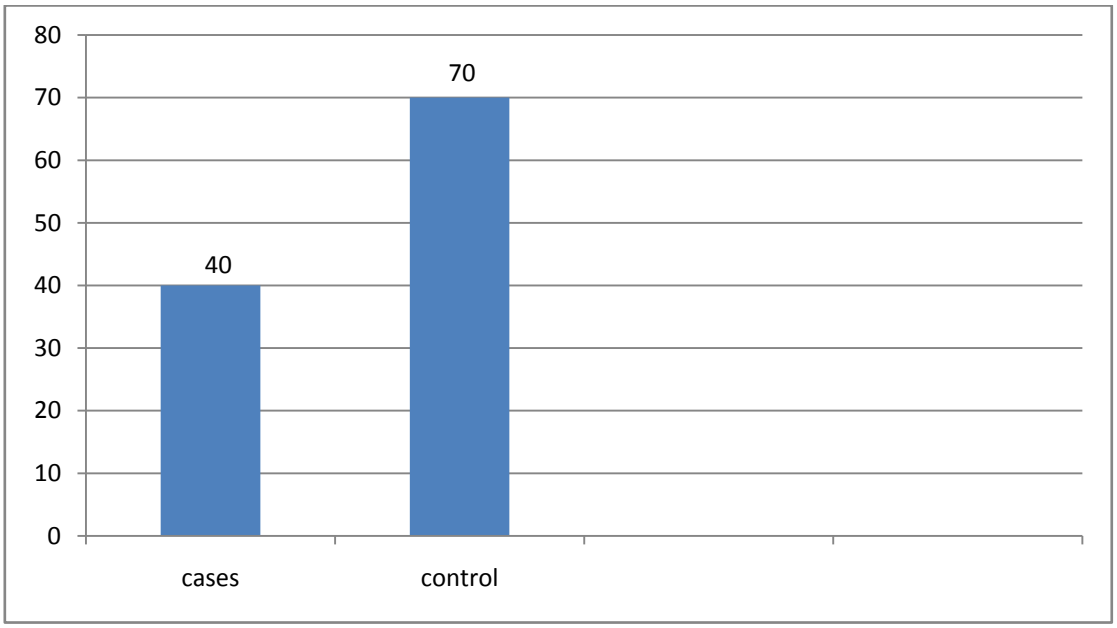
COMPARISON OF BLOOD LOSS FROM THE END OF SURGERY TO TWO HOURS POSTPARTUM

Blood loss (ml)	cases			Control			Mean	P value
End of surgery to 2 hrs post op	max	min	median	max	Min	median	30	<0.001
	50	30	40	120	50	70		

Thus table 3 reveals to us that the median blood loss from time of end of caesarean to two hours post operative period in study group is 40 ml compared to70ml in the control group with the mean difference of 30 ml with highly significant statistical difference among blood loss in both study and control group . Thus,patients on tranexemic acid had 30 ml of blood loss less compared to that of control group who did not receive tranexemic acid.

GRAPH 2

BAR DIAGRAM SHOWING COMPARISON OF BLOOD LOSS FROM END OF SURGERY TO 2 HOURS POSTPARTUM



Graph demonstrates the amount of blood loss from the end of surgery to two hours postpartum among both cases and control.

TABLE 4

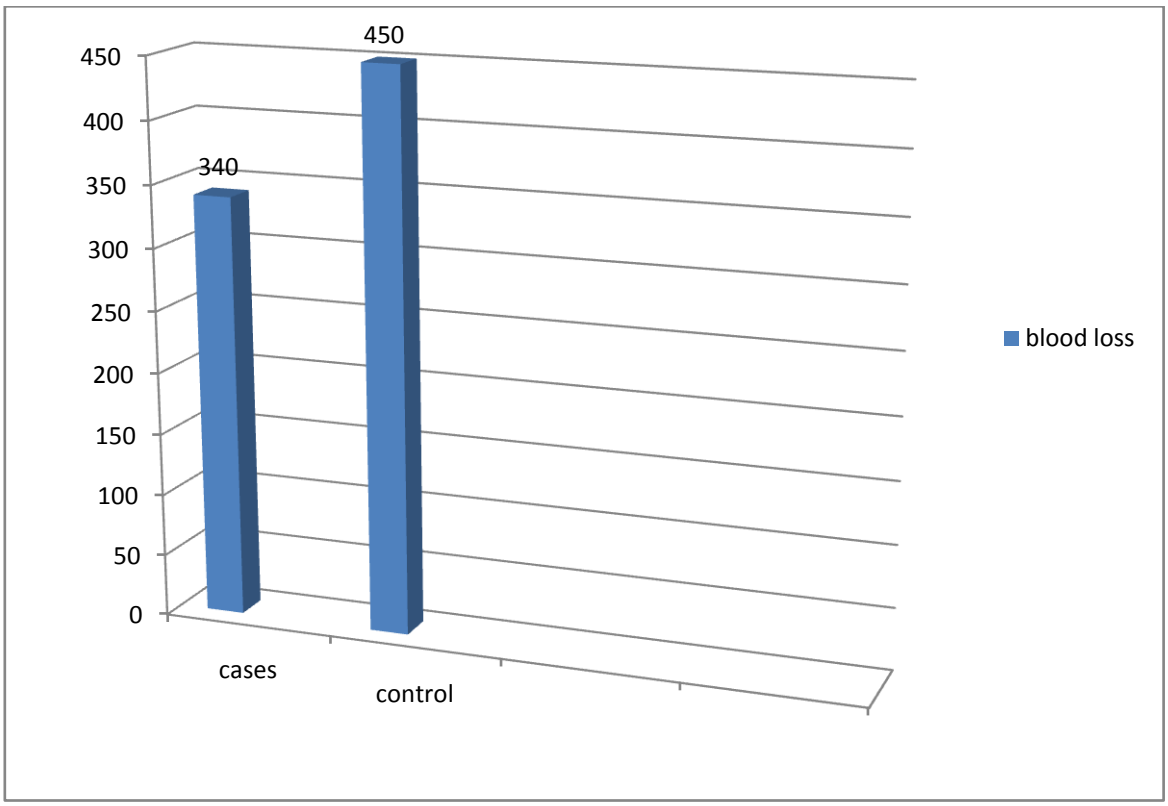
COMPARISON OF TOTAL BLOOD LOSS FROM THE END OF PLACENTAL DELIVERY TO TWO HOURS POSTPARTUM

Blood loss	cases (median)	control (median)	Mean difference	P value
Total(ml)	340	450	110	<0.001

Thus the blood loss starting right from the end of caesarean section to that of two hours of postpartum shows of median of 340 ml blood lossin cases and 450 ml blood loss in control group. Thus accounting for 110 ml of blood loss less in the patients receiving tranexemic acid as such which the p value of <0.001which shows that it is highly significant statistical difference in blood loss among both study and control group. Thus patients who receive tranexemic acid before surgery has 110ml less blood loss compared to the patients who did not receive tranexemic acid.

GRAPH 3

BAR DIAGRAM SHOWING TOTAL BLOOD LOSS FROM THE END OF PLACENTAL DELIVERY TO THE END OF TWO HOURS POST PARTUM



The graph demonstrates the total blood loss from the end of placental delivery to two hours post partum.

TABLE 5

COMPARISON OF BLOOD LOSS MORE THAN 500 ML AMONG BOTH THE GROUPS

Blood loss>500ml	cases		control		P value
present	0	0	7	9.33%	0.013
Absent	75	100%	68	90.67%	
Total	75	100 %	75	100%	

Table 5 shows incidence of blood loss more than 500ml among both cases and control .the p value obtained here is 0.013 which was statistically significant. The incidence of PPH is more in the control group compared to the study group which had no incidence of PPH

GRAPH 4

BAR DIAGRAM SHOWING COMPARISON OF BLOOD LOSS MORE THAN 500 ml AMONG BOTH THE GROUPS

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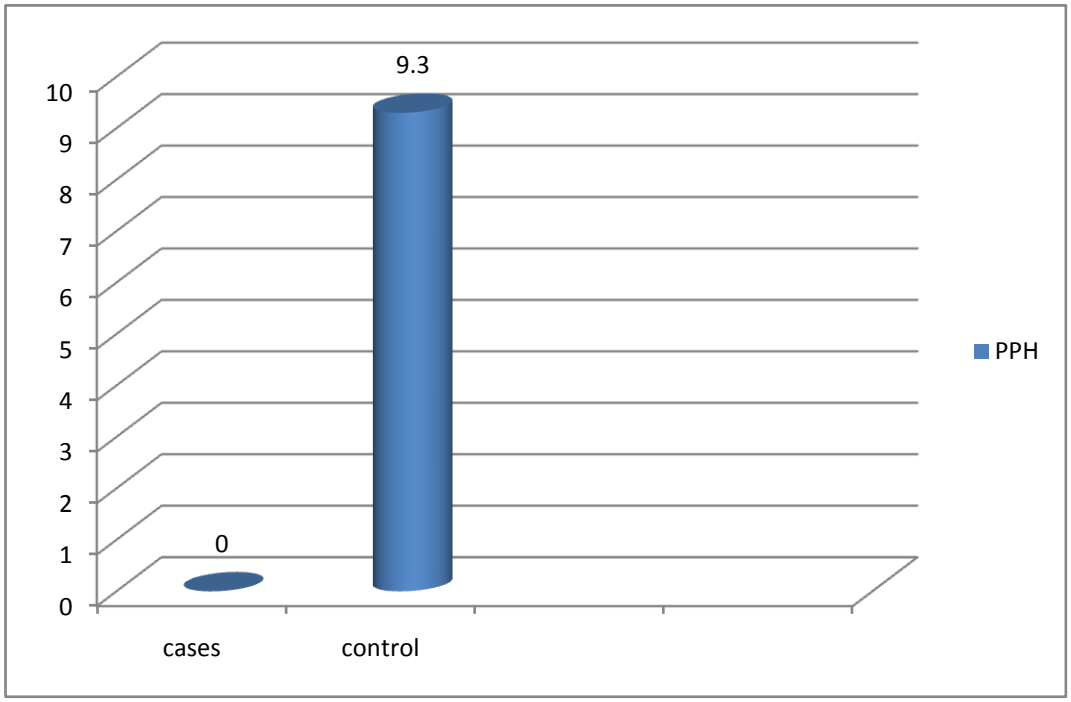


The graph demonstrates the incidence of PPH among both the groups which is obviously nil in the study group.

TABLE 6

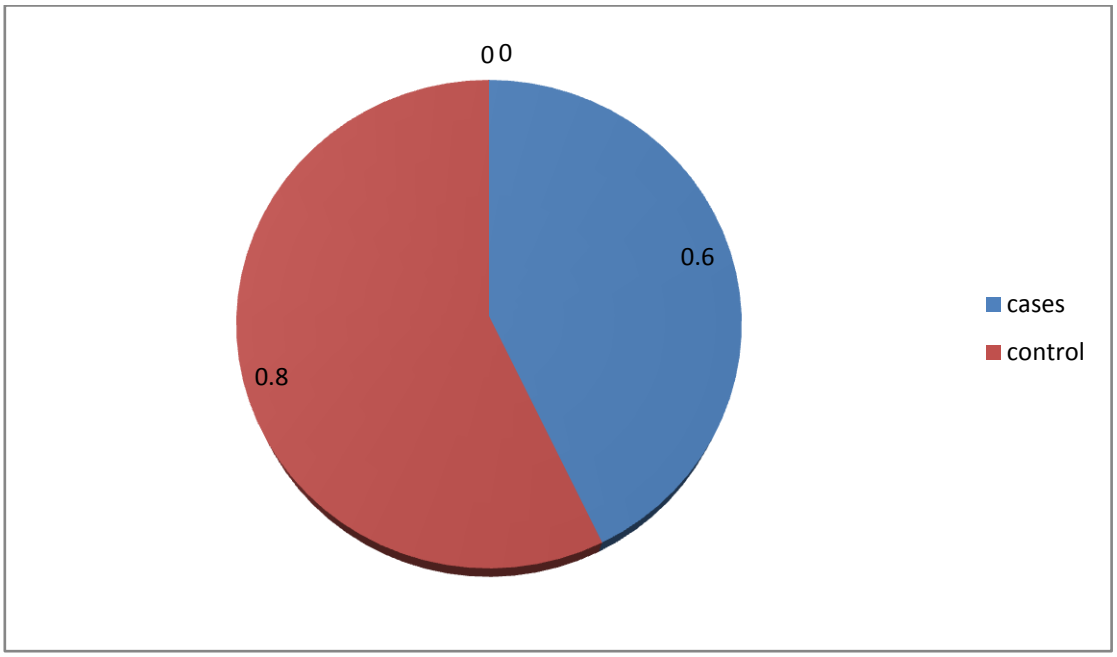
COMPARISON OF THE DIFFERENCE IN HAEMOGLOBIN % POST OPERATIVELY AMONG BOTH THE GROUPS

HB	cases			control			Mean difference	P value
After surgery	maximum	minimum	median	Max	min	median	0.20	<0.001
	1.2	0.2	0.60	3.9	0.2	0.80		

This Table 6 shows the Hb% difference which is given in median for the group. Given tranexemic acid came as 0.60 and for the group not given tranexemic acid came as 0.80 and the difference in Hb % between two groups is 0.20 with p value of <0.001 which is highly significant statistically.

GRAPH 5

PIE CHART SHOWING THE DIFFERENCE OF HEMOGLOBIN AMONG BOTH THE GROUPS



This graph demonstrate the mean hemoglobin difference is more in the control group. The hemoglobin difference is less among study group in the post operative period.

TABLE 7

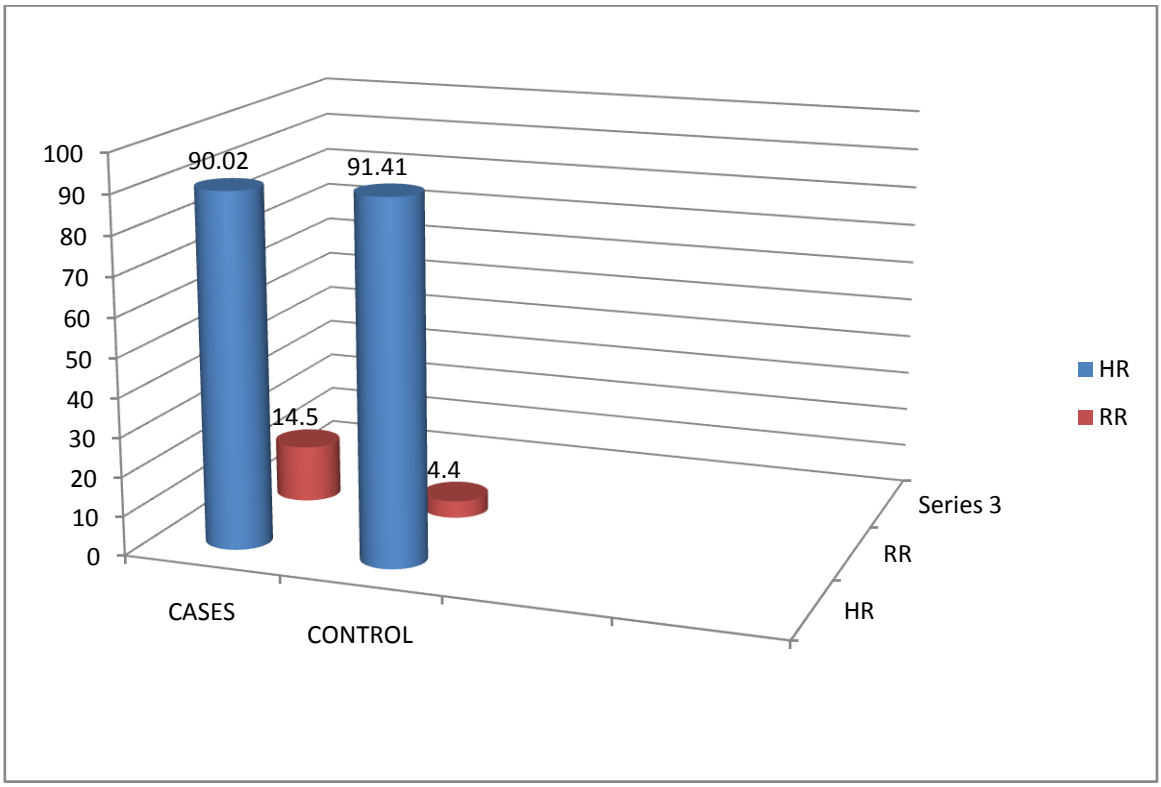
COMPARISON OF VITAL SIGNS AMONG BOTH GROUPS – POST OPERATIVELY

Vital signs	Cases		control		P value
	mean	SD	mean	SD	
HR	90.02	4.23	91.41	3.86	0.038
RR	14.5	1.45	15.16	1.62	0.014

Table 7 shows that heart rate (HR) and respiratory rate (RR) in both the groups does not show statistical significant difference.

FIGURE 6

BAR DIAGRAM SHOWING COMPARISON OF VITAL SIGNS AMONG BOTH THE GROUPS



Coming on to effect of respiratory rate and heart rate , there is no statistical significance among both the groups.

TABLE 8

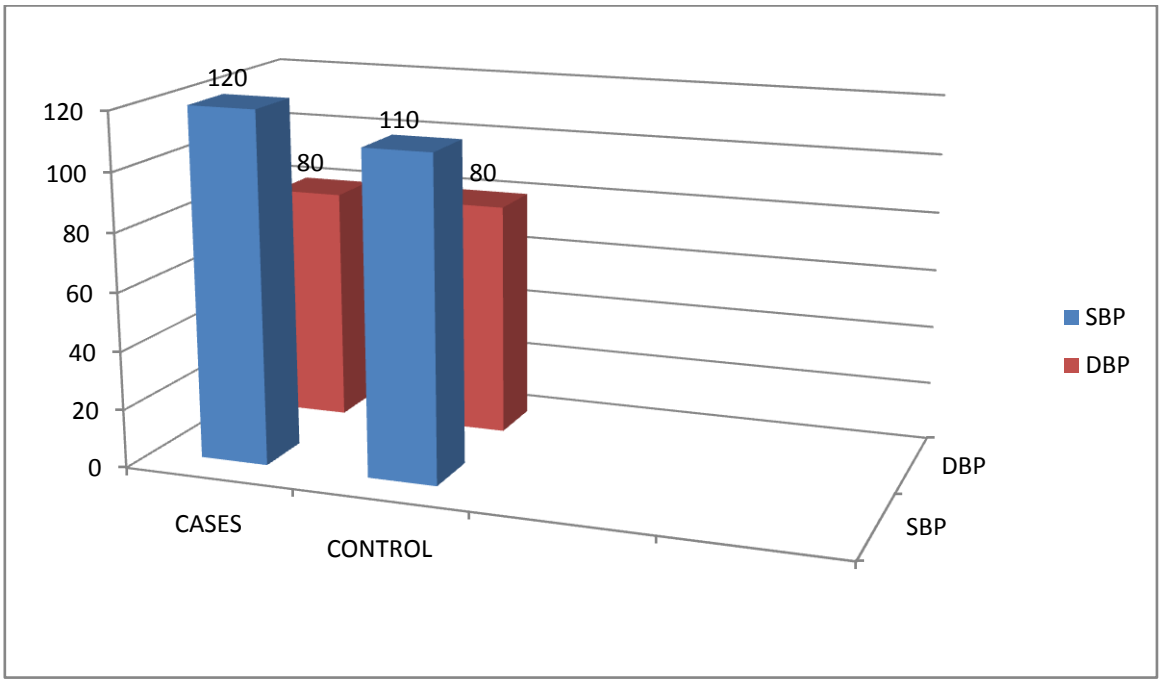
COMPARISON OF BLOOD PRESSURE MEASUREMENT AMONG BOTH THE GROUPS

BP		STUDY		CONTROL		P VALUE
		N	%	N	%	
SBP	100	15	20	15	20	0.858
	110	33	44	36	48	
	120	27	36	24	32	
DBP	60	0	0	6	8	0.040
	70	33	44	28	37.33	
	80	42	56	41	54.67	

Thus the systolic blood pressure (SBP) and diastolic blood pressure (DBP) does not show any significant difference between both the groups.

TABLE8

BAR DIAGRAM SHOWING COMPARISON OF BLOOD PRESSURE AMONG BOTH THE GROUPS



Among both the groups there is no significant difference in both systolic and diastolic blood pressure intra operatively as well post operatively.

TABLE 9

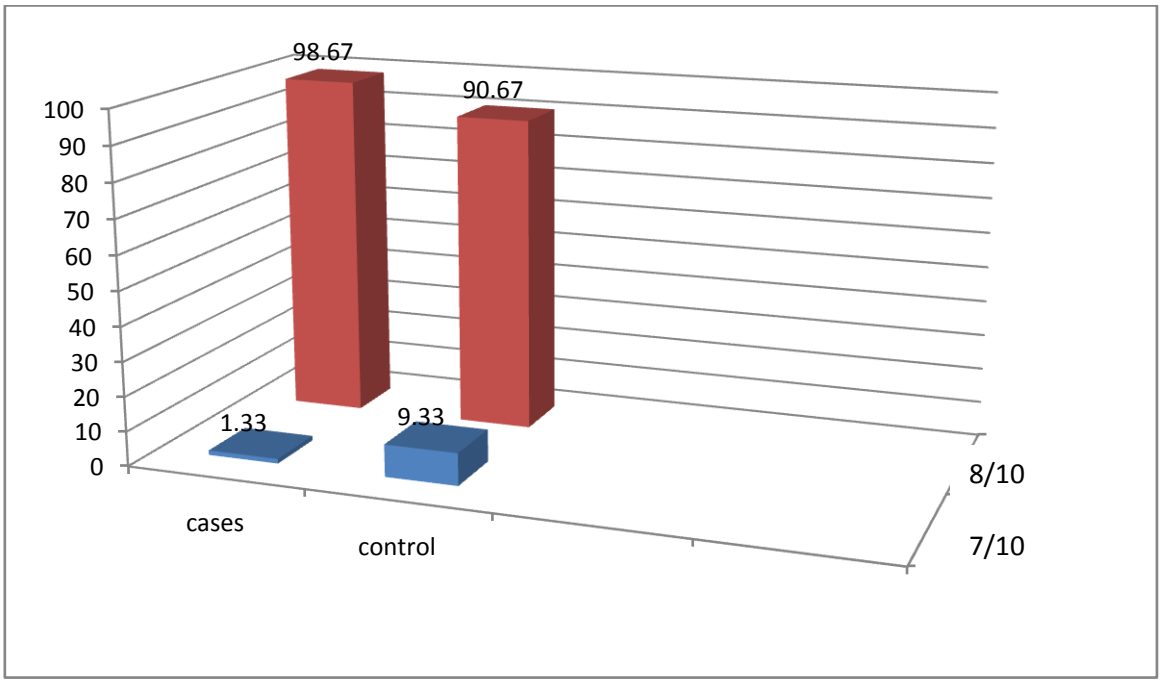
COMPARISON OF APGAR SCORE AMONG BOTH THE GROUPS

APGAR SCORE	CASES		CONTROL		P VALUE
	N	%	N	%	
7/10	1	1.33	7	9.33	0.29
8/10	74	98.67	68	90.67	

Table 9 reveals that apgar score at 1 minute in newborn in both the groups as the p value as 0.29 which is not satistically significant.

FIGURE 8

BAR DIAGRAM SHOWING COMPARISON OF APGAR SCORE AMONG BOTH THE GROUPS



There is no obvious variation in the apgar score among both the groups. The neonatal outcome remains the same among both the groups.

TABLE 10

COMPARISON OF ADVERSE DRUG REACTION IN BOTH THE GROUPS

ADVERSE REACTIONS	CASES		CONTROL		P VALUE
	N	%	N	%	
NAUSEA	15	54.55	9	52.94	0.934
VOMITING	12	45.45	8	47.06	
OTHERS	-	-	-	-	-
THROMBOSIS	-	-	-	-	

FIGURE 9

BAR DIAGRAM SHOWING ADVERSE EFFECTS AMONG BOTH THE GROUPS

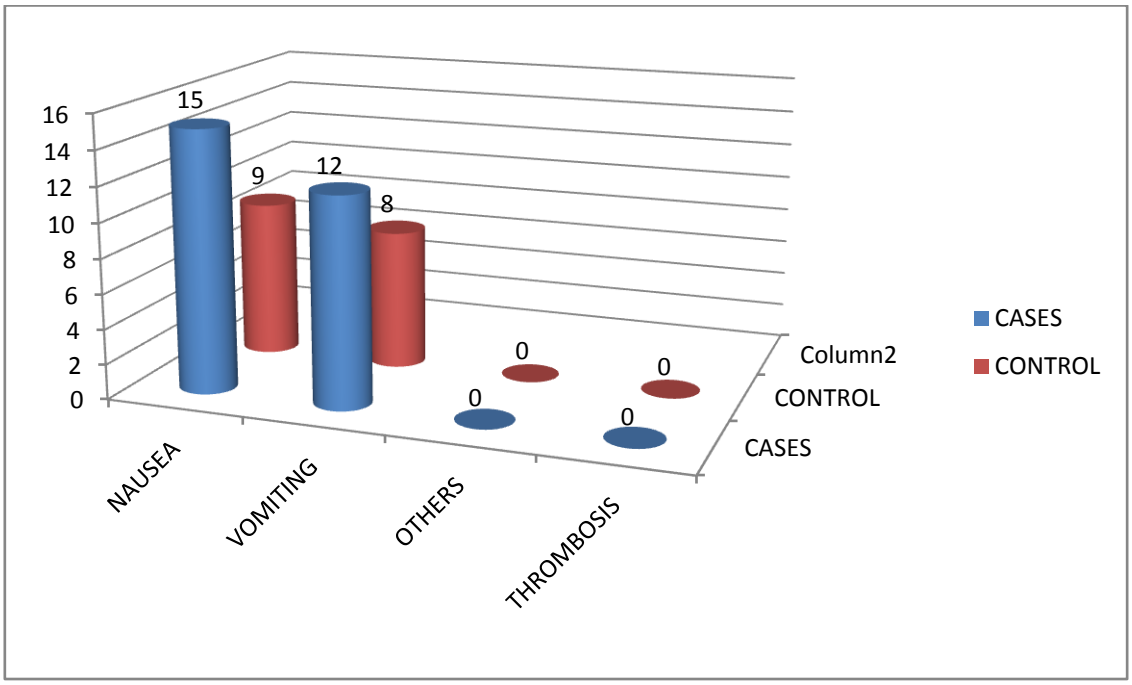


Table 10. shows the adverse side effects in the study group of administration of tranexemicacid has nausea and vomiting among 15 and 12 subjects respectively. No other complications as well as no evidence of thrombosis in this study group. Whereas in control group the incidence of nausea and vomiting is 9 and 8 respectively. No other significant adverse effect seen. This gives the p value of 0.934 which is not statistically significant . Thus it can be concluded that the incidence of side effects profile is not increased among the tranexemic acid group compared to the control where tranexemic acid is not given. As there is no incidence of

thrombosis among both the groups ,the fear of main side effect of tranexemic acid as thrombosis can be left apart according to this present study.

DISCUSSION

Tranexemicacid is an anti fibrinolytic agent. Tranexemic acid literally dismiss the dissolution of hemostatic fibrin by plasmin due to its anti fibrinolytic effect. More over during the placental delivery the fibrinogen and fibrin gets degraded rapidly and simultaneously plasminogen activator as well as fibrin degradation products increases as the fibrinolytic system gets activated. It is found that this fibrinolytic activity which has started immediately after placental delivery can last for nearly 6 -8 hours post partum resulting in more blood loss.Thus,taking this into account,we have decided to take tranexemic acid which is an anti fibrinolytic agent for our study.

Tranexemic acid actively binds at the lysine receptor binding site of fibrin and hence it stabilizes fibrin and prevents it undergoing fibrinolysis. Tranexemic acid also inhibits plasminogen conversion into plasmin by inhibiting plasminogen activator . Thus tranexemic acid is used in treatment of reducing blood loss for many years.

From our study it has been evident that tranexemic acid significantly reduces the blood loss from the time of placental delivery to that of 2 hours postpartum..The values that are being obtained in the study group is 340 ml and in the control group is 450 ml with a median blood loss of 110 ml

which gives the p value of <0.001 which is highly satistically significant difference. Thus, the blood loss is nearly 35 % less than in the control group which is significant. The blood loss from the time of placental delivery to end of caesarean section is 350 ml in study group and 380 ml in control group giving p value as <0.001 which is statistically significant.

The blood loss from the end of caesarean section to two hours postpartum as given in the study group is 40 ml and in the control group is 70 ml with a p value of <0.001 which is once again statistically significant.

Thus, tranexemic acid administration reduces the blood loss right from the end of placental delivery to two hours postpartum. In case of calculating blood loss >500 ml in both the groups gives us the value as follows. There is no evidence of blood loss >500 ml in the study group where as the incidence of blood loss more than 500 ml in control group is 7 giving us the impression that blood loss is more in the group who are not administered tranexemic acid prior to surgery and the p value that is obtained is 0.013 which is significant satistically. The list extends that the group has been administered tranexemic acid showed the post operative hemoglobin difference less compared to the group not administered with tranexemic acid with p value of <0.001. hence the efficacy of using tranexemic acid is proven as it not only reduces blood loss and decreases

incidence of post partum haemorrhage as well as improves the hemoglobin difference.

Coming on to vital parameters both the study and control group does not show any statistical difference in heart rate, respiratory rate, blood pressure. Hence tranexemic acid can be safely given without any significant changes in vital parameters.

In view of Apgar score administration of tranexemic acid has no effects on outcome difference in the Apgar among both the group.In view of adverse effects among the tranexemic acid administered group does not show any significant difference compared with that of control group. There is no evidence of increased risk of thrombosis in the present study.

Afshan shahid and Ayesha khan conducted a randomized double blind placebo controlled trial and concluded that tranexemic acid significantly reduced the amount of blood loss during the lower segment caesarean section but it did not reduce the blood loss significantly after the caesarean section. Its use was not associated with any side effects or complications like thrombosis. Tranexemic acid can be safely and effectively used in women who are undergoing lower segment caesarean segment in order to reduce intra operative blood loss.

Anne sophie ducloy bouthors et al conducted a study to determine administration of high dose of tranexemic acid at the time of diagnosis of post partum hemorrhage Could reduce blood loss and finally demonstrate that high dose of tranexemic acid can reduce blood loss and maternal morbidity in women with post partum hemorrhage. The side effects observed in this study was only mild and transient. However further study is required to investigate further about the action of tranexemic acid in reducing post partum hemorrhage.

Gohe l Mayur, patel purvi et al conducted a study to know the efficacy and safety of tranexemic acid in reducing blood loss during and after the lower segment caesarean section and concluded that tranexemic acid significantly reduces the amount of blood loss during and after caesarean section and they are not associated with any side effects or any complications and can be safely used in caesarean section.

Panagio tespeitsidis et al conducted similar study to evaluate the efficacy and safety of tranexemic acid in the management of obstetric hemorrhage, so that tranexemic acid can be used for prevention as well as treatment for postpartum hemorrhage and concluded that the tranexemic acid is highly effective in reducing the amount of blood loss after delivery which may be either caesarean section or vaginal delivery as well as decreases the need for blood transfusion.

Haleemashakur et al famous THE WOMEN trial (World Maternal Anti fibrinolytic trial) and the main aim here is intention to treat basis which showed significant reduction in blood loss.

H. Abdel Aleem et al conducted similar study to evaluate the effectiveness of tranexemic acid in reducing blood loss in elective caesarean section and found positive results as it may be of much benefits for anaemic women as well as women who needs blood transfusion.

Sekhavat et al concluded the same results as tranexemic acid statistically reduces blood loss from the end of caesarean section to two hours post partum and its use is not associated with any side effects or complications.

TarabrinGalich has done a similar study to evaluate blood loss during caesarean section with tranexemic acid and found that blood loss is significantly reduced to nearly 60 %.

Mehmet B Senturk et al conducted a study to assess the efficacy and safety of an intravenous tranexemic acid to reduce bleeding during child birth and concluded and confirmed that tranexemic acid is effective in reducing blood loss with no worries about thrombosis.

CONCLUSION

1. Tranexemic acid is highly effective in significantly reducing the blood loss during as well as after the lower segment caesarean section.
2. Tranexemic acid usage has not resulted in excess blood loss exceeding more than 500 ml and hence the incidence of post partum hemorrhage among the tranexemic acid group is minimal.
3. Tranexemic acid administration is not associated with any adverse drug reactions.
4. There is no risk of thrombosis among the users of tranexemic acid.
5. Fetal outcome as given by apgar score is not affected by the usage of tranexemic acid.

SUMMARY

This study was carried out to find whether the usage of tranexemic acid prior to surgery to all patients undergoing elective lower segment caesarean section in Coimbatore medical college hospital during the period of August 2014 to August 2015 has resulted in reduction in blood loss during and after caesarean section

1. The confounding factor age is comparable in both the groups.
2. Study group showed marked decrease in amount of blood loss from the time of placental delivery to the end of caesarean section around 80 ml compared to that of control group.
3. Similar scenario is that the blood loss from the end of caesarean section to two hours postpartum in the study group is 40 ml less than in the control group.
4. The amount of blood loss from the time of placental delivery to that of two hours postpartum among the study group is 110ml less compared to that of control group.
5. Tranexemic acid has significant difference in hemoglobin difference post operatively as blood loss is decreased compared to study group.
6. There is no significant difference in vital parameter in both the groups.

7. There is no significant difference in Adverse effects in both the groups.
8. There is no significant difference in Apgar score in newborn in both the groups.

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PROFORMA

NAME IP.NO

AGE DOA

OCCUPATION DOD

ADDRESS

DIAGNOSIS

PRESENTING COMPLAINTS

Amenorrhea

Labour pains : yes /no

Leaking per vagina : yes /no

Bleeding per vagina : yes /no

Appreciates foetal movements well : yes /no

History suggestive of pre eclampsia : yes /no

History suggestive of anaemia : yes /no

MENSTRUAL HISTORY:

Age of menarche:

Last menstrual period:

Expected date of delivery:

Cycles : regular /irregular

MARITAL HISTORY:

Married since:

Consanguious /non consanguious marriage:

OBSTETRIC HISTORY;

Gravida

Para

Living

Abortion

HISTORY OF PREVIOUS PREGNANCY:

PAST HISTORY:

Hypertension yes/no

Diabetes yes/no

Bronchial asthma yes/no

Tuberculosis yes/no

Epilepsy yes /no

Rheumatic heart disease yes/no

Hypothyroid yes/no

Previous blood transfusion

PERSONAL HISTORY:

Diet:

Appetite:

Bladder/bowel habbits:

GENERAL EXAMINATION

Build:

Nourishment:

Pallor:

Pedal edema:

Height:

Weight:

BMI:

Pulse rate:

Blood pressure:

Breast:

Spine:

Thyroid:

SYSTEMIC EXAMINATION

Cardiovascular system:

Respiratory system:

Central nervous system:

Obstetric examination:

Inspection :

Palpation:

Auscultation:

Per vaginal examination:

INVESTIGATIONS

BLOOD HB

URINE ROUTINE

HIV

HBsAg

RBS

Blood urea

Serum creatinine

Liver function test:

Coagulation profile:

Indication of caesarean section:

Time of givingtranexemic acid:

Time of skin incision:

Baby details:

Single term alive ---------- baby

Birth weight:

Time:

Apgar score at 1 min: at 5 min:

After delivery of placenta:

Dry mop weight: (A)

After soakage weight(B)

Total blood loss: (B) - (A) =QUANTITY OF BLOOD LOSS :

MASTER CHART WITH TRANEXEMIC ACID

vitals																						
							Blood Loss (ml)			HB % (gm/dl)			BP									
S. No.	NAME	IP No	Age (years)	Parity	Time of giving tranexemic acid	Time of giving skin incison	Placental delivery to end of CS	End of CS to 2 hrs Postpartum	Total	Before Delivery	24 Hrs After delivery	Fall in Hb% (gm/dl)	Before Delivery SBP(mm of Hg)	After Delivery SBP(mm of Hg)	Before Delivery DBP(mm of Hg)	After Delivery DBP(mm of Hg)	HR	RR	APGAR	Use of additional oxytocins	Complication	Side effect
1	Aparna	10264	26	G2P1L1	9.20am	9.40am	300	40	340	10.2	10	0.2	120	110	80	70	88	16	8/10	-	-	-
2	Shakila banu	11157	22	G2P1L1	9.30am	9.50am	250	40	290	11.4	10.8	0.6	11o	120	80	80	84	14	8/10	-	-	-
3	Mallika	10258	33	G2P1L1	9.25am	9.41am	280	40	320	10.4	9.8	0.6	120	110	70	80	88	16	8/10	-	-	Nausea
4	shubha	10412	24	G2P1L1	9.05am	9.26am	300	40	340	11.8	11.2	0.6	110	110	80	70	90	15	8/10	-	-	-
5	Kalaivani	13767	25	G2P1L1	10.45am	11.00am	280	50	330	10.2	10	0.2	120	120	70	70	82	14	8/10	-	-	Vomiting
6	Raabi	17430	23	G2P1L1	9.30am	9.50am	300	40	340	10.4	10	0.4	120	110	70	80	86	16	8/10	-	-	-
7	Saranya	18327	25	G2P1L1	9.40am	10.00am	280	40	320	10.8	10	0.8	110	100	80	80	92	15	8/10	-	-	Nausea
8	Victoria	18317	21	G2P1L1	9.10am	9.27am	300	30	330	11	10.8	0.2	120	120	80	70	84	16	7/10	-	-	-
9	Shajira	19165	28	G3P2L2	9.25am	9.42am	280	50	330	10.8	10.4	0.4	110	100	80	80	90	15	8/10	-	-	-
10	Gowri maheshwari	20864	28	G2P1L1	10.05am	10.28am	250	50	300	10.8	10.2	0.6	110	120	70	70	80	12	8/10	-	-	-
11	Punitha	22481	29	G3P1L1A1	10.25am1	10.42am	300	40	340	10	9.8	0.2	110	100	80	70	86	16	8/10	-	-	-
12	Saranya	21448	25	G2P1L1	9.35am	9.55am	300	30	330	12	11.4	0.6	120	120	70	80	88	14	8/10	-	-	-
13	Kani mozhi	24208	28	G2P1L1	10.25am	10.41am	280	40	320	10.2	9.8	0.4	110	110	80	80	82	2	8/10	-	-	Vomiting
14	Rekha	24839	21	G2P1L1	10.35am	10.57am	300	40	340	10	9.8	0.2	110	120	80	70	88	16	8/10	-	-	Nausea
15	Eswari	25531	33	G4P1L1A2	9.30am	9.47am	280	50	330	10.8	10	0.8	120	110	70	80	90	14	8/10	-	-	-
16	Subha	26364	24	G2P1L1	9.30am	9.45am	300	40	340	10.4	10.2	0.2	110	120	80	80	86	15	8/10	-	-	-
17	Sunitha	26157	29	G3P1L1A1	9.05am	9.27am	280	40	320	10	9.4	0.6	120	100	80	80	90	12	8/10	-	-	Vomiting
18	Susila	26175	28	G2P1L1	9.00am	9.22am	300	30	330	10	9.8	0.2	110	110	70	80	92	16	8/10	-	-	-
19	Saranya	27213	22	G2P1L1	9.10am	9.30am	320	40	360	10	9.2	0.8	110	120	80	70	90	14	8/10	-	-	Vomiting
20	Menaga	27265	29	G2P1L1	10.10am	10.25am	300	40	340	11.2	10.4	0.8	110	120	70	70	84	15	8/10	-	-	-

21	Nirmala	28280	28	G2P1L1	11.25am	11.38am	280	50	330	10.8	10.4	0.4	120	110	80	70	94	12	8/10	-	-	-
22	Kalaivani	28549	21	G2P1L1	9.25am	9.37am	280	40	320	11.2	10.6	0.6	120	110	80	80	88	16	8/10	-	-	Nausea
23	Saraswathi	27610	20	G2P1L1	9.20am	9.37am	300	30	330	10.8	10	0.8	110	100	70	70	92	12	8/10	-	-	-
24	Sakina	30228	30	G3P2L2	9.15am	9.30am	300	40	340	10	9.8	0.2	120	110	80	80	94	14	8/10	-	-	-
25	Nirmala	29641	20	G3P1L1A1	9.30am	9.43am	300	40	340	10.4	9.8	0.6	110	100	80	70	82	12	8/10	-	-	-
26	Vennila	30440	26	G2P1L1	9.30am	9.47am	280	40	320	10.8	10	0.8	100	110	80	80	88	14	8/10	-	-	Nausea
27	Valli	31728	26	G2P1L1	9.30am	9.45am	300	30	330	10.4	10.2	0.2	110	120	70	80	86	14	8/10	-	-	-
28	Rabiya	31294	24	G2P1L1	9.30am	9.50am	320	40	360	10.4	9.8	0.6	120	110	80	80	96	15	8/10	-	-	Vomiting
29	Priya	33052	24	G2P1L1	9.15am	9.33am	300	30	330	10.2	9.6	0.8	110	100	70	70	86	16	8/10	-	-	-
30	Sangeetha	34682	25	G2P1L1	9.25am	9.40am	320	30	350	11.4	10.8	0.6	120	120	70	80	90	14	8/10	-	-	Nausea
31	Ranganayaki	33782	22	G2P1L1	10.30am	10.50am	300	40	340	12	11.4	0.6	110	120	80	70	94	12	8/10	-	-	Vomiting
32	Saranya	35701	27	G3P2L1	10.45am	11.04am	280	50	330	13	12.8	0.2	120	110	70	80	84	16	8/10	-	-	-
33	Pauvul rani	37045	31	G2P1L1	9.30am	9.50am	300	40	340	12	11.2	0.8	110	120	80	70	90	14	8/10	-	-	-
34	Saradha	36868	35	G3P2L2	10.30am	10.52am	300	50	350	9.8	9	0.8	100	110	70	70	98	16	8/10	-	-	Nausea
35	Manju	37346	25	G2P1L1	9.15am	9.32am	280	50	330	10.4	10	0.4	120	110	80	80	94	12	8/10	-	-	-
36	Priyanka	36104	24	G2P1L1	9.20am	9.47am	300	30	330	10.8	10	0.8	110	110	70	70	86	16	8/10	-	-	Vomiting
37	Sujitha	37082	33	primi	9.15am	9.33am	300	40	340	12	11.6	0.4	120	100	80	70	90	16	8/10	-	-	-
38	Vigini	38262	25	G2P1L1	9.10am	9.25am	320	30	350	10	9.4	0.6	110	120	70	80	96	14	8/10	-	-	-
39	Jerina begum	32824	35	G2P1L1	9.50am	10.10am	300	40	340	10	9.2	0.8	120	110	80	70	94	16	8/10	-	-	-
40	Kalaivani	36849	29	G2P1L1	10.10am	10.28am	280	50	330	10.2	10	0.2	110	110	70	80	90	14	8/10	-	-	-
41	Nirmala	37526	24	G2P1L1	9.25am	9.45am	300	303	330	10.8	0.2	0.6	120	100	80	70	98	16	8/10	-	-	Nausea
42	Radhika	39479	25	G2P1L1	10.40am	11.05am	310	40	350	10.8	9.8	1	110	100	80	80	90	12	8/10	-	-	-
43	Sindhu	37879	27	G3P1L1A1	9.50am	10.12am	300	30	330	12	11.4	0.6	110	120	70	80	86	16	8/10	-	-	Nausea
44	Muruga lakshmi	39680	21	primi	10.15am	10.35am	280	40	320	10.8	10	0.8	120	110	80	80	82	14	8/10	-	-	-
45	Shanthi	39291	27	G3P1L1A1	9.10am	9.33am	300	40	340	10.8	9.8	1	110	110	80	70	90	15	8/10	--	-	-
46	Meena	61159	25	G2P1L1	9.05am	9.23am	300	30	330	11	10.8	0.2	120	110	70	70	88	12	8/10	--	-	-
47	Priyadharshini	62484	27	G2P1L1	9.20am	9.40am	300	40	340	10.9	10	0.9	1101	100	80	70	90	14	8/10	-	-	Nausea

48	Ranjitha	62896	23	G2P1L1	9.25am	9.47am	320	30	350	10.6	10.4	0.2	120	110	70	80	92	16	8/10	-	-	-
49	Pushpa	59439	26	G2P1L1	9.45am	10.05am	300	40	340	11	10.1	0.9	110	120	80	90	90	14	8/10	-	-	Vomiting
50	Valiammal	64252	33	G2P1L1	9.10am	9.33am	280	50	330	11.8	10.6	1.2	120	110	80	70	94	16	8/10	-	-	-
51	Jaya lakshmi	64630	33	G5P1L1A3	9.00am	9.18am	300	40	340	10.8	10	0.8	110	120	70	80	90	12	8/10	-	-	-
52	Mariammal	64414	27	G2P1L1	10.00am	10.23am	320	30	350	12.8	12	0.8	120	110	80	70	88	14	8/10	-	-	Vomiting
53	Meghala	58624	23	G2P1L1	9.50am	10.12am	300	40	340	11	10.8	0.2	110	120	70	80	92	16	8/10	-	-	-
54	Aswathi	66782	18	primi	10.00am	10.20am	280	50	330	12.4	11.4	1	120	110	80	70	92	14	8/10	-	-	Nausea
55	Poongodi	67753	29	G2P1L1	9.35am	9.56am	300	30	330	10.81	10	0.8	110	120	70	80	90	16	8/10	-	-	-
56	Manju	68030	24	G2P1L1	9.25am	9.43am	300	40	340	10.8	10	0.8	110	120	80	70	88	16	8/10	-	-	Nausea
57	Vanitha	67794	23	G2P1L1	10.45am	11.08am	310	40	350	11	10.4	0.6	120	100	70	80	90	14	8/10	-	-	-
58	Lokamani	70415	35	G2P1L1	9.25am	9.42am	290	40	330	10.4	9.8	0.6	110	100	80	70	94	16	8/10	-	-	-
59	Nadhiya	70622	27	G2P1L1	9.30am	9.47am	300	30	330	10.8	10.4	0.4	120	110	70	80	90	12	8/10	-	-	Vomiting
60	Lokanayaki	71805	22	G2P1L1	12.05pm	12.23pm	280	40	320	13.2	12.8	0.4	110	110	80	70	98	14	8/10	-	-	-
61	Ramya	72111	23	G2P1L1	9.25am	9.40am	300	40	340	12.4	11.8	0.6	120	120	70	80	96	16	8/10	-	-	-
62	Sathya	72912	20	G2P1L1	9.15am	9.33am	310	30	340	11	0.8	0.2	100	110	80	80	92	14	8/10	-	-	Nausea
63	Mariammal	73837	32	G2P1L1	9.30am	9.47am	300	50	350	12.4	11.8	0.6	110	120	70	80	90	16	8/10	-	-	-
64	Renuga devi	76040	25	G3P1L1A1	9.45am	10.05am	310	30	340	10.9	10.4	0.5	120	110	80	70	94	14	8/10	-	-	-
65	Sathya	75867	23	G2P1L1	10.45am	11.05am	300	40	340	10.8	10	0.8	110	120	70	80	90	16	8/10	-	-	
66	Sudha	76928	24	G3P1L1A1	9.10am	9.28am	300	30	330	11.8	10.9	0.9	120	110	70	80	94	15	8/10	-	-	-
67	Manjula	77380	33	G2P1L1	9.25am	9.42am	280	50	330	9.8	9.4	0.4	110	120	80	70	98	14	8/10	-	-	-
68	Suseela banu	76930	27	G2P1L1	10.10am	10.28am	300	30	330	11.4	10.8	0.6	120	110	80	80	90	16	8/10	-	-	Vomiting
69	Ramya	78139	24	G2P1L1	9.10am	9.33am	290	40	330	10.4	10	0.4	110	120	70	80	96	16	8/10	-	-	Nausea
70	Rajathi	78091	25	G2P1L1	9.15am	9.38am	310	30	340	12.2	11.8	0.4	120	110	80	70	92	14	8/10	-	-	-
71	Uma maheshwari	80225	33	G3P1L1A1	9.00am	9.20am	300	40	340	12	11.4	0.6	110	120	80	80	86	16	8/10	-	-	-
72	Indira	79375	27	G5P1L1A3	9.55am	10.16am	280	50	330	10.4	10	0.4	120	110	70	80	90	14	8/10	-	-	Nausea
73	Velumani	80421	24	G2P1L1	9.10am	9.29am	300	40	340	11.2	10.8	0.4	110	120	80	70	88	16	8/10	-	-	-
74	Selvi	80931	27	G2P1L1	9.10am	9.29am	280	40	320	13	12.2	0.8	120	100	70	80	94	12	8/10	-	-	-
75	Kalavathi	80549	36	G2P1L1	9.25am	9.43am	300	30	330	12.8	11.9	0.9	110	100	80	80	98	14	8/10	-	-	Vomiting

MASTER CHART WITHOUT TRANEXEMIC ACID

vitals																						
							Blood Loss (ml)			HB % (gm/dl)			BP									
S. No.	NAME	IP No	Age (years)	Parity	Time of giving tranexemic acid	Time of giving skin incison	Placental delivery to end of CS	End of CS to 2 hrs Postpartum	Total	Before Delivery	24 Hrs After delivery	Fall in Hb% (gm/dl)	Before Delivery SBP(mm of Hg)	After Delivery SBP(mm of Hg)	Before Delivery DBP(mm of Hg)	After Delivery DBP(mm of Hg)	HR	RR	APGAR	Use of additional oxytocins	Complication	Adverse Effects
1	Namagiri	11212	28	G3P1L0A1	-	9.38am	380	50	430	12	11	1	110	110	80	70	86	14	8/10	-	-	-
2	Kalpana	12072	24	G2P1L1	-	9.30am	360	70	430	12.2	11.4	0.8	120	110	80	80	88	18	8/10	-	-	-
3	Sumathi	12503	27	G2P1L0	-	9.33am	380	80	460	11.8	10.4	1.4	110	100	80	70	90	16	7/10	-	-	-
4	Shanthi	16503	21	G2P1L1	-	11.52am	350	70	420	11.8	10.8	1	120	110	70	80	96	14	8/10	-	-	-
5	Balamani	18316	25	G2P1L1	-	9.47am	380	70	450	12.2	11.4	0.8	110	120	80	70	94	12	8/10	-	-	Nausea
6	Vimala	19116	26	G2P1L1	-	10.43am	390	60	450	13.2	11.2	2	120	100	70	80	98	14	8/10	-	-	-
7	Najimunisha	19150	28	G3P2L2	-	9.33am	400	70	470	12.8	10.9	1.9	110	120	80	70	96	16	8/10	-	-	-
8	Manju	18481	21	G3P2L2	-	10.35am	390	50	440	14	12.4	1.6	120	110	80	70	90	14	8/10	-	-	-
9	Umavathi	20053	23	G2P1L1	-	9.40am	900	120	520	13.4	9.8	3.6	120	100	80	60	98	18	7/10	oxytocin	PPH	-
10	Maharnisha	23899	23	G2P1L1	-	10.40am	430	70	500	12.8	11.8	1	110	120	70	70	90	16	8/10	-	-	-
11	Jaffar nisha	24007	27	G2P1L1	-	9.37am	400	60	460	12.4	11.8	0.6	120	110	80	80	92	14	8/10	--	-	-
12	Radha	25582	31	G2P1L1	-	10.17am	380	80	460	11	10.2	0.8	110	120	70	80	96	16	8/10	-	-	vomiting
13	Vijayalakshmi	24855	34	G2P1L1	-	11.10am	380	60	440	10.8	9.6	1.2	120	110	80	80	88	15	8/10	-	-	-
14	Jayashree	26481	22	G2P1L1	-	10.43am	350	80	430	11	10.2	0.8	110	120	80	80	92	12	8/10	-	-	-
15	Saranya	27213	22	G2P1L1	-	9.33am	380	70	450	11.8	11	0.8	120	110	70	80	94	14	8/10	-	-	-
16	Ranjitha	25890	21	G2P1L1	-	9.33am	400	60	460	12.8	12.2	0.6	110	120	80	70	92	16	8/10	-	-	Vomiting
17	Samsath begum	28563	21	G2P1L1	-	9.37am	350	60	410	12	10.8	1.2	110	110	70	80	88	15	8/10	-	-	-
18	Krishna veni	27090	21	G2P1L1	-	9.42am	360	80	440	10	9.4	0.6	120	110	80	80	94	14	8/10	-	-	-
19	Priya	29942	29	G2P1L1	-	9.50am	380	60	440	10.4	9.8	0.6	110	120	70	80	98	16	8/10	-	-	vomiting
20	Kalpana	30678	28	G2P1L1	-	9.38am	400	70	470	11	10.2	0.8	120	110	80	70	92	14	7/10	-	-	-

21	Ushana begum	31480	26	G2P1L1	-	9.47am	370	80	450	11.2	10.4	0.8	110	120	70	80	90	16	8/10	-	-	nausea
22	Revathi	30947	28	G2P1L1	-	11.02am	400	60	460	11.8	10	1.8	120	110	80	70	94	14	8/10	-	--	-
23	Parvathi	31340	35	G2P1L1	-	11.12am	950	100	1050	13.9	10	3.9	110	100	70	60	92	16	8/10	oxytocin	PPH	-
24	Pushparani	32479	29	G3P1L1A1	-	10.15am	380	80	460	10.4	9.2	1.2	120	110	80	80	98	14	8/10	-	-	-
25	Laila	33492	28	G2P1L1	-	10.29am	400	70	470	12	11.2	0.8	100	110	60	60	96	16	8/10	-	-	-
26	Saranya	35701	27	G3P2L1	-	11.05am	380	80	460	10.8	9.8	1	110	120	70	70	86	16	8/10	--	-	nausea
27	Sujitha	34422	26	G2P1L1	-	12.20pm	400	80	480	12	11.2	0.8	120	110	80	70	96	15	8/10	-	-	-
28	Divya	36523	33	G4P1L1A2	-	10.18am	380	70	450	11	10.6	0.4	110	110	70	80	90	18	8/10	-	-	-
29	Sheela devi	36538	32	G2P1L1	-	9.33am	390	60	450	12.2	11..6	0.6	120	110	80	70	92	16	8/10	-	-	nausea
30	Kala	34711	29	G2P1L1	-	11.00am	360	70	430	10.2	9.4	0.8	120	110	80	80	88	14	8/10	-	-	vomiting
31	Nandhini	37099	20	G2P1L1	-	9.50am	400	50	450	10.8	10.4	0.4	1001	110	80	80	94	16	8/10	-	-	-
32	Jeeva rani	36604	23	G2P1L1	-	10.43am	920	90	1010	12	9.6	2.4	120	100	80	60	86	16	8/10	oxytocin	PPH	-
33	Saraswathi	32264	22	G2P1L1	-	10.43am	380	60	440	11.2	10.6	0.6	110	120	70	80	88	14	8/10	-	-	-
34	Uma maheshwari	37823	26	G3P1L1A1	-	10.33am	380	70	450	12	11.4	0.6	120	110	80	70	90	15	8/10	-	-	-
35	Visalatchi	38909	24	G2P1L1	-	9.47am	350	80	430	10	9.2	0.8	110	120	70	80	92	14	8/10	-	-	nausea
36	Nandhini	40529	24	G2P1L1	-	9.43am	400	60	460	10.4	9.8	0.6	110	120	80	80	90	16	8/10	-	-	-
37	Nagarani	60798	33	G3P2L1	-	9.38am	380	70	450	11.8	11	0.8	120	110	70	80	92	14	8/10	-	-	-
38	Lurth preetha	62435	26	G2P1L1	-	10.24am	370	70	440	12	11.2	0.8	110	120	80	70	90	18	8/10	-	-	-
39	Aymona beevi	63647	26	G2P1L1	-	11.02am	420	60	480	10.4	9.6	0.8	120	110	70	80	94	16	8/10	-	-	-
40	Dhanalakshmi	63959	28	G3P1L1A1	-	9.33am	400	80	480	9.8	9	0.8	110	110	70	70	84	14	8/10	-	-	-
41	Kalamani	63937	28	G4P1L1A2	-	10.20am	370	70	340	10.8	10	0.8	120	100	80	70	90	16	8/10	-	-	vomiting
42	Anisha	63863	20	PRIMI	-	9.23am	360	80	340	11	10	1	110	120	70	80	92	14	8/10	-	-	-
43	Mariammal	64667	25	G3P2L1	-	9.47am	380	80	460	12	11.4	0.6	120	110	80	80	90	16	8/10	-	-	-
44	Sujitha	65585	27	G2P1L1	-	10.30am	400	60	460	11	10.2	0.8	110	100	70	70	92	18	8/10	-	-	vomiting
45	Anushya	65330	25	G2P1L1	-	11.18am	380	70	450	11.2	10.8	0.4	120	110	80	70	90	16	8/10	-	-	-
46	Krishna veni	68385	30	G2P1L1	-	9.38am	400	70	470	12	10.8	1.2	100	110	80	80	92	14	8/10	-	-	-
47	Ponnammal	69003	32	G4P1L1A2	-	9.25am	380	60	440	14	13	1	110	120	70	80	90	12	8/10	-	-	-

48	Radha mani	68388	22	G2P1L1	-	10.17am	370	70	440	13	12.2	0.8	100	110	70	70	86	14	8/10	-	-	-
49	Gayathri	68890	21	G2P1L1	-	10.08am	380	60	440	10	9.4	0.6	110	100	80	80	84	16	8/10	-	-	vomiting
50	Manimeghalai	68033	19	PRIMI	-	9.43am	400	60	460	11.8	10.4	1.4	120	110	70	70	80	14	7/10	-	-	-
51	Selvi	70677	26	G2P1L1	-	9.47am	380	70	450	10.2	9.8	0.4	110	120	80	80	84	14	7/10	-	-	-
52	Rekha	71383	23	G2P1L1	-	11.13am	400	60	460	10.8	10	0.8	120	110	70	80	86	16	8/10	-	-	-
53	Vanitha	70841	20	G2P1L0	-	9.47am	380	70	450	11	10.2	0.8	110	120	80	70	86	14	8/10	-	-	nausea
54	Usha	70857	26	G3P1L1A1	-	11.33am	380	60	440	10.2	9	1.2	120	110	80	80	86	16	8/10	-	-	-
55	Durga devi	72801	24	G2P1L1	-	10.25am	940	80	1020	11	8.8	2.2	110	100	70	60	98	20	8/10	oxytocin	PPH	-
56	Kaliya devi	74140	25	G2P1L1	-	9.43am	400	50	450	12	11.4	0.6	110	120	70	70	90	14	8/10	-	-	-
57	Vigneshwari	73179	19	PRIMI	-	9.57am	380	60	440	12.8	11	1.8	120	110	80	70	92	16	8/10	-	-	-
58	Karthiga	74052	23	G2P1L1	-	11.48am	390	60	350	12.2	10.2	2	110	120	70	70	94	16	8/10	-	-	nausea
59	Arul mozhi	74598	31	G2P1L1	-	9.29am	940	80	1020	13	12.2	0.8	120	100	80	60	96	20	8/10	oxytocin	PPH	-
60	Madhavi	74605	25	G3P2L2	-	9.38am	400	60	460	12	11.2	0.8	110	120	70	80	94	16	8/10	-	-	-
61	Sathya	72479	24	G2P1L1	-	10.08am	380	70	450	10	9	1	120	110	80	80	96	16	8/10	-	-	-
62	Sapna	71755	24	G2P1L1	-	9.35am	850	110	960	11	10.2	0.8	110	100	70	80	94	14	8/10	oxytocin	PPH	-
63	Muneeshwari	76313	29	G2P1L1	-	9.40am	370	70	440	12	11	1	120	110	76	80	90	15	8/10	-	-	-
64	Benazir	76557	25	G2P1L1	-	9.30am	380	70	450	13	12.8	0.2	110	110	70	80	92	16	8/10	-	-	-
65	Poongodi	76556	32	G3P2L2	-	9.38am	380	80	460	12	11.2	0.81	120	100	80	80	90	14	8/10	-	-	vomiting
66	Anitha	53932	23	G2P1L1	-	9.43am	400	50	450	11	9.8	1.2	100	110	80	80	92	16	8/10	-	-	-
67	Saradha	54508	25	G3P1L1A1	-	9.45am	370	50	420	12.4	11.8	0.6	110	100	70	80	94	14	8/10	-	-	nausea
68	Jothi mani	53379	26	primi	-	9.22am	400	60	460	12	10.6	1.4	110	120	80	80	90	12	8/10	-	-	-
69	Preetha	53842	30	G3P1L1A1	-	9.20am	380	60	440	13	12	1	100	110	70	80	92	14	8/10	-	-	
70	Malathi	52442	22	G3P1L1A1	-	9.23am	960	80	1040	11.8	10	1.8	110	100	80	70	98	16	7/10	Oxytocin	PPH	-
71	Mano ranjitham	47212	28	G4P1L1A2	-	9.28am	350	90	440	11.2	11	0.2	120	110	70	70	92	14	8/10	-	-	-
72	Jameela	47212	28	G2P1L1	-	10.27am	360	80	440	12.2	11.6	0.6	100	120	80	80	90	16	8/10	-	-	nausea
73	Amirthajothi	49681	23	G3P2L1	-	9.27am	370	80	450	11.4	10.8	0.6	110	120	70	70	96	14	8/10	-	-	-
74	Thamarai	50106	20	G2P1L1	-	9.37am	400	50	450	11.8	11	0.8	120	120	80	70	94	12	8/10	-	-	-
75	Bhuvaneshwari	51718	20	G2P1L1	-	10.45am	410	70	480	12.4	11.6	0.8	110	100	70	80	90	16	7/10	-	-	-

KEY TO MASTER CHART

LSCS	Lower Segment Caesarean Section
BL	Blood Loss
HR	Heart Rate
RR	Respiratory Rate
BP	Blood Pressure
SBP	Systolic blood Pressure
DBP	Diastolic Blood Pressure
G	Gravid
P	Para
A	Abortion
L	Live birth
Primi	Primi gravida
Hb	Hemoglobin

INFORMED CONSENT FORM

TITLE OF THE TOPIC:

"**EVALUATION OF EFFECTIVENESS OF PROPHYLACTIC PARENTERAL TRANEXEMIC ACID IN REDUCING BLOOD LOSS DURING AND AFTER ELECTIVE LOWER SEGMENT CESAREAN SECTION** "

A RANDOMIZED CASE CONTROLLED PROSPECTIVE STUDY

PURPOSE OF RESARCH

I have been informed that this study will test the efficacy of tranexamic acid in reducing blood loss during and after cesarean section. No side effects were noted in mother as well as in neonate.

PROCEDURE

I understand that I will be assigned a group of patients for LSCS.I will be examined and a series of questions will be asked by resarcher. My history and physical finding will be recorded. My results will be evaluated in a systemic way. I will not be asked for any follow up.

RISK AND DISCOMFORTS

I understand that I may have some discomfort due to IV administration of tranexamic acid .There are no major risks involved and side effects are minimal.

BENEFITS

I understand that my participation in the study will have direct benefit to me, it is mainly designed to decreases the blood loss during and after LSCS.

CONFIDENTIALITY

I understand that medical information produced by this study will become part of hospital record and will be subject to confidentiality record and privacy regulation of Coimbatore Medical College & Hospital.

REQUEST FOR MORE INFORMATION

I understand that I may ask more questions about the study at any time and understand that I will be informed of any significant new finding discovered during the course of this study which might influence my continued participation.

If during the study or later I wish to discuss my participation or concerns regarding this study with a person not directly involved I am aware that the other staff members are available to talk with me.

The copy of this consent form will be given to me to keep for careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION

I understand that my participation is voluntary and that I may refuse to participate or withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care in the hospital and also understand that the researcher may terminate my participation in the study if at any time she feels the need and explain me the reason to do so and help to arrange for my further appropriate treatment

INJURY STATEMENT

I understand that in the unlikely event or any injury due to my participation in the study will be reported promptly, then medical treatment will be available to me but no further compensation would be provided by the hospital. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to Mrs. _____ the purpose of the research, the procedures required and the possible risks and benefits to the best of ability.

Investigator:

Date:

I confirm that the researcher has explained to me the purpose of research the study procedures that I will undergo and the possible risks and discomforts as well as benefits that I may experience. Alternatives to my participation in the study have also been discussed. I have read and I understand this consent form. Therefore, I agree to give my consent to participate as a subject in this research project.

Participant:

Date:

ஒப்புதல் படிவம்

பெயர் :

வயது :

பாலினம் :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் மகப்பேறு மற்றும் பெண்கள் மருத்துவ துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவி **ச.கோ. விஜய்ஸ்ரீ** அவர்கள் மேற்கொள்ளும் **"அறுவை சிகிச்சை பிரசவத்தின் போதும் அதன் பின்னர் ஏற்படும் இரத்த இழப்பைக் குறைப்பதில் டீரனசிமிக் அமிலத்தின் திறன் - ஒரு மதிப்பீடு"** பற்றிய ஆய்வில் செய்முறை மற்றும் அனைத்து விளக்கங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த அய்வில் என்னைப் பற்றிய அனைத்து விவரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்வில் இருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம்

தேதி கையொப்பம் / ரேகை

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